Int J Clin Exp Pathol 2014;7(2):808-814
www.ijcep.com /ISSN:1936-2625/IJCEP1312050

Case Report

Concomitant occurrence of IgG4-related pleuritis and periaortitis: a case report with review of the literature

Mitsuaki Ishida[1,2], Keiko Hodohara[3], Aya Furuya[3], Aya Fujishiro[3], Hiroko Okuno[1], Miyuki Yoshii[1], Akiko Horinouchi[1], Ayaka Shirakawa[1], Ayumi Harada[1], Muneo Iwai[2], Keiko Yoshida[2], Akiko Kagotani[2], Takashi Yoshida[1], Hidetoshi Okabe[1,2]

[1]Department of Clinical Laboratory Medicine, [2]Division of Diagnostic Pathology, [3]Department of Hematology, Shiga University of Medical Science, Shiga, Japan

Received December 17, 2013; Accepted January 3, 2014; Epub January 15, 2014; Published February 1, 2014

Abstract: IgG4-related sclerosing disease is an established disease entity with characteristic clinicopathological features. Some recent reports have demonstrated that this disease can occur in the respiratory system including the pleura. Herein, we describe the first documented case of concomitant occurrence of IgG4-related pleuritis and periaortitis. A 71-year-old Japanese female with a history of essential thrombocythemia presented with persistent cough and difficulty in breathing. Computed tomography demonstrated thickening of the right parietal pleura, pericardium, and periaortic tissue and pleural and cardiac effusions. Histopathological study of the surgical biopsy specimen of the parietal pleura revealed marked fibrous thickening with lymphoplasmacytic infiltration. Phlebitis was noted, however, only a few eosinophils had infiltrated. Immunohistochemical study revealed abundant IgG4-positive plasma cell infiltration and high ratio of IgG4-/IgG-positive plasma cells (84%). Therefore, a diagnosis of IgG4-related pleuritis was made with consideration of the elevated serum IgG4 level (684 mg/dL). Recently, the spectrum of IgG4-related sclerosing disease has expanded, and this disease can occur in the pleura, pericardium, and periaortic tissue. Although histopathological analysis of the pericardium and periaortic tissue was not performed in the present case, it was suspected that thickening of the pericardium and periaortic tissue was clinically due to IgG4-related sclerosing disease. Our clinicopathological analyses of IgG4-related pleuritis and pericarditis reveal that this disease can present as dyspnea and pleural and pericardial effusion as seen in the present case, therefore, it is important to recognize that IgG4-related sclerosing disease can occur in these organs for accurate diagnosis and treatment.

Keywords: IgG4-related sclerosing disease, pleuritis, periaortitis

Introduction

IgG4-related sclerosing disease is an established systemic fibroinflammatory disease characterized clinically by the formation of tumor-like lesions and elevated serum IgG4 concentration [1-3], and histopathologically by the presence of fibrosclerosis and dense lymphoplasmacytic infiltration with abundant IgG4-positive plasma cells and high IgG4/IgG-positive plasma cell ratio accompanied by eosinophilic infiltration and obliterative phlebitis [2-5]. This disease entity was first established in sclerosing pancreatitis (autoimmune pancreatitis) [1]. Since then, it has been recognized that this disorder can occur in various organs including the liver, bile duct, gallbladder, nasal cavity, salivary gland, lacrimal gland, aorta, kidney, pituitary gland, and retroperitoneum [4-20]. Recently, this disease was also demonstrated in the respiratory system [21-26]. Taniguchi *et al*. reported the first documented case of interstitial pneumonia associated with IgG4-related autoimmune pancreatitis, and they demonstrated an infiltration of IgG4-positive plasma cells in the alveolar septum [21]. Zen *et al*. reported that inflammatory pseudotumor (plasma cell granuloma) of the lung shows the same histopathological and immunohistochemical features of IgG4-related sclerosing pancreatitis, and this condition is also included in the spectrum of IgG4-related sclerosing disease [22]. Subsequently, Shrestha *et al*. analyzed the histopathological features of

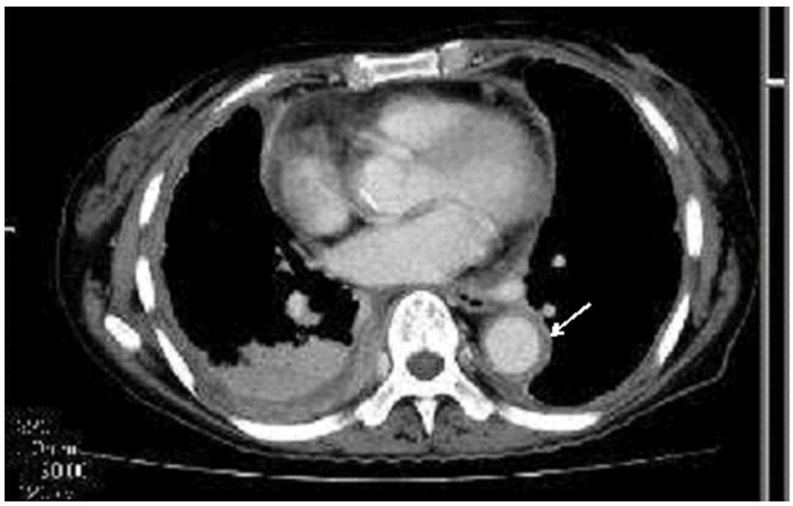

Figure 1. Contrast-enhanced computed tomography showing thickening of the right pleura, pericardium, and periaortic tissue (arrow), as well as pleural and cardiac effusions.

the pulmonary lesions of 6 cases with autoimmune pancreatitis [23]. They demonstrated that these lesions were characterized by the presence of lymphangitic distribution of plasma cell-rich inflammatory infiltration, active fibrosis, remarkable intimal inflammation involving both pulmonary veins and arteries, fibrinous pleuritis, and dense peribronchial inflammation [23]. Zen *et al*. also analyzed the clinicopathological features of 16 cases of IgG4-related pulmonary disease and 5 cases of pleural lesions, and showed that three of 5 cases of pleural IgG4-related disease had extrapulmonary IgG4-related lesions [24], however, concomitant occurrence of periaortitis has not been reported yet. In this report, we document the first case of concomitant IgG4-related pleuritis and periaortitis, and review the literature.

Case report

A 71-year-old Japanese female, who had never smoked, presented with persistent cough and difficulty in breathing on exertion. She had been suffering from thrombocytosis for approximately 20 years, which was diagnosed as essential thrombocythemia 5 years earlier. She had been administered aspirin and hydroxycarbamide for essential thrombocythemia. A contrast-enhanced computed tomography demonstrated thickening of the right parietal pleura and pericardium, pleural effusion in the right thoracic cavity, and cardiac effusion (**Figure 1**). Thickening of the periaortic tissue from the aortic arch to the thoracic aorta was also detected, which was suggestive of periaortitis (**Figure 1**). Mild mediastinal lymph node swelling was also observed. Neither tumorous lesions nor features suggestive of interstitial pneumonia were present in the lung. She had no clinical history of pancreatitis,cholangitis,allergic diseases, and asthma.

Laboratory tests revealed anemia, thrombocytosis, and elevated soluble interleukin-2 receptor (red blood cells 2.89 × 10^{12}/L (range 3.8-4.8), hemoglobin 8.1 g/L (11.3-15.0), white blood cells 14.9 × 10^{9}/L (3.0-8.0), eosinophils 2.6% (<7%), platelets 1047 × 10^{9}/L (150-400), soluble interleukin-2 receptor 3,170 U/mL (135-483). Serum IgG was slightly elevated (1,756 mg/dL (range 870-1,700)), and IgG4 level was elevated (684 mg/dL (4.8-105)).

She underwent surgical biopsy of the right parietal pleura to confirm the diagnosis.

Histopathological study demonstrated marked fibrous thickening of the parietal pleura (**Figure 2A**, **2B**). Dense fibrosis was observed, which extended into the fatty tissue (**Figure 2A**). Lymphoplasmacytic infiltration was present within the fibrous lesion (**Figure 2A**, **2B**), and small lymphoid follicles were also observed. Lymphocytes were small in size and plasma cells also appeared mature (**Figure 2C**). Although typical obliterative phlebitis was not noted, phlebitis with subendothelial lymphoplasmacytic infiltration was observed (**Figure 2C**, inset). Only a few eosinophils had infiltrated into the lesion (1-2 eosinophils/10 high power fields).

Immunohistochemical and *in situ* hybridization studies were performed using an autostainer (Ventana) by the same method as previously reported [27-32]. Many CD138-positive plasma cells had infiltrated into the lesion (**Figure 3A**), as well as relatively abundant CD3-positive T-lymphocytes, and a few CD20-positive B-cells. There was also abundant IgG4- and

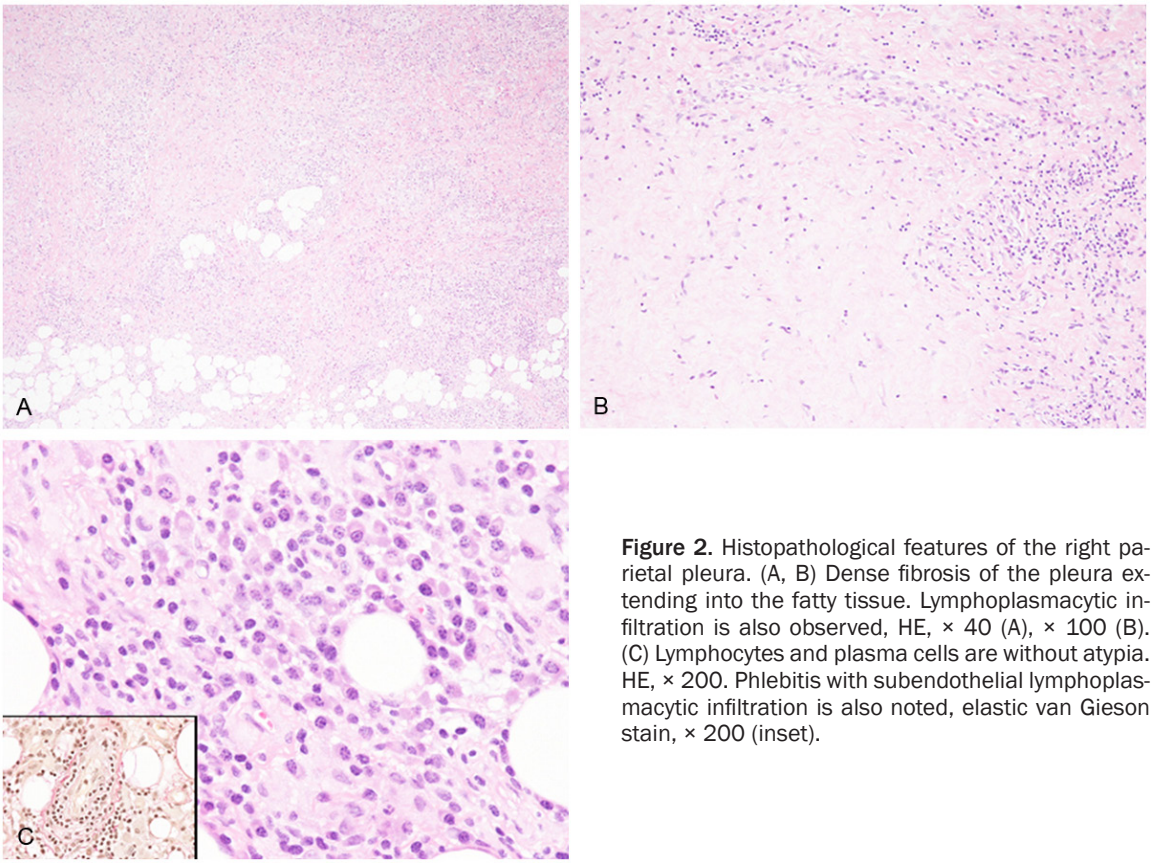

Figure 2. Histopathological features of the right parietal pleura. (A, B) Dense fibrosis of the pleura extending into the fatty tissue. Lymphoplasmacytic infiltration is also observed, HE, × 40 (A), × 100 (B). (C) Lymphocytes and plasma cells are without atypia. HE, × 200. Phlebitis with subendothelial lymphoplasmacytic infiltration is also noted, elastic van Gieson stain, × 200 (inset).

IgG-positive plasma cells infiltration (**Figure 3B**, **3C**). The IgG4-positive plasma cell count was 273/10 high-power fields and the ratio of IgG4-/IgG-positive plasma cells was 84%. *Kappa*- and *lambda* chain-positive plasma cells were evenly distributed by *in situ* hybridization analyses. Moreover, no EBER-positive cells were detected by *in situ* hybridization.

Accordingly, an ultimate diagnosis of IgG4-related pleuritis was made. Although no biopsy or surgical resection of the pericardium and aorta was performed, the thickening of the pericardium and periaortic tissue was suspected to be clinically due to IgG4-related sclerosing disease.

Thus, steroid therapy (40 mg/day) was administered, which immediately improved the cough and difficulty in breathing. Follow-up computed tomography revealed a mild decrease in the pleural thickening, and the pleural and cardiac effusions were no longer observed.

Discussion

IgG4-related sclerosing disease has been recognized as a distinct clinicopathological disease entity, which has been demonstrated to occur in various organs [4, 6]. Recently, this disease has been shown to also involve the respiratory system [21-26], and that IgG4-related lung lesions can occur in the form of tumorous lesions or interstitial pneumonia [21-25]. Moreover, some recent reports revealed that IgG4-related sclerosing disease can occur in the pleura. **Table 1** summarizes the clinicopathological features of the previously reported cases of IgG4-related pleuritis as well as the present one. Zen *et al*. reported a series of 5 cases of IgG4-related pleuritis [24], and the remaining cases were single case reports [25, 26, 33-35]. IgG4-related sclerosing disease frequently affected middle-aged males (average 62.7 years, range from 29 to 78 years; male: female 8: 3), however, this disease can also occur in young female (29-years-old) [35]. The most common complaint was dyspnea, and pleural thickening and effusion were the most frequent signs of this disease. Mediastinal lymph node swelling and lung nodules were observed in 3 cases. Pericardial thickening and effusion, and pericarditis were observed only in the present case, although 5 cases had non-

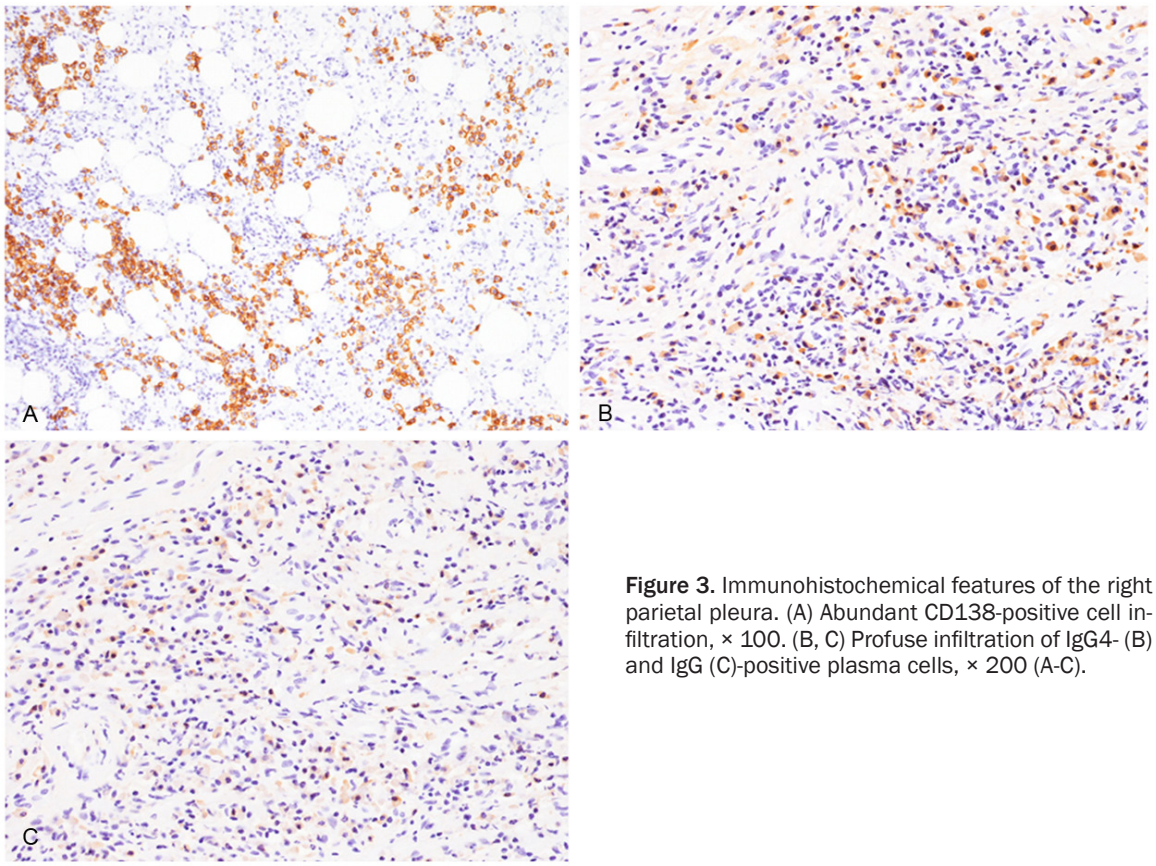

Figure 3. Immunohistochemical features of the right parietal pleura. (A) Abundant CD138-positive cell infiltration, × 100. (B, C) Profuse infiltration of IgG4- (B) and IgG (C)-positive plasma cells, × 200 (A-C).

Table 1. Clinicopathological features of IgG4-related pleuritis

Case No.	Age	Gender	Initial presentation	Location	Associated diseases	Serum IgG-4 (mg/dL)	Steroid treatment	Reference
1	65	Male	Pleural effusion and swelling of the mediastinal lymph nodes	Left pleural effusion, lung nodules, and mediastinal lymph node swelling.	Mikulicz's disease	1,194	Effective	[33]
2	63	Female	Dyspnea	Bilateral pleural and pericardium effusions with pericardial thickening. Mediastinal lymph node swelling.	Autoimmune pancreatitis, Hashimoto thyroiditis	420	Effective	[34]
3	74	Male	Dyspnea	Pleural effusion, nodular lesion in the lung, as well as extensive pleural thickening and adhesion.	None	Not available	Not available	[25]
4-8	49-76	Males	Not available	Pleura in 4 cases, lung and pleura in 1 case.	3/5 cases (+).	Not available	Not available	[24]
9	78	Male	General fatigue and fever	Bilateral pleural effusion and thickening.	None	483	Not performed	[26]
10	29	Female	Chest pain and dyspnea	Bilateral pleural thickening and effusion	None	136	Effective	[35]
Present case	71	Female	Cough and difficulty in breathing	Pleural thickening and effusion, pericardial thickening and effusion, and mediastinal lymph node swelling.	None	684	Effective	

pleural and non-pulmonary IgG4-related sclerosing diseases. Steroid therapy was effective in all cases, in whom information regarding therapeutic effects was available. The characteristic histopathological features of IgG4-related pleuritis are as follows: i) the pleura is severely thickened by diffuse sclerosing inflammation, and extends into the subpleural fibrous and fatty tissue, ii) inflammation consists of lymphocytes and plasma cells, iii) eosinophilic infiltration is frequently seen, but neutrophilic infiltration is rare, iv) obliterative phlebitis is

commonly found, and v) abundant IgG4-positive plasma cell infiltration is observed, and the ratio of IgG4-/IgG-positive plasma cells is high (>40%) [24]. Although only a small number of eosinophils had infiltrated and typical obliterative phlebitis was not present (but phlebitis with subendothelial lymphoplasmacytic infiltration was noted) in the present case, the remaining typical features were observed. Therefore, an ultimate diagnosis of IgG4-related pleuritis was made with consideration of the elevated serum IgG4 (684 mg/dL).

In 2008, Kasashima *et al*. reported a close relationship between IgG4-related sclerosing disease and inflammatory abdominal aortic aneurysm, and they concluded that inflammatory abdominal aortic aneurysm can be classified as IgG4-related and non-IgG4-related [11]. Subsequently, it has been demonstrated that IgG4-related inflammatory aortic aneurysm can occur in the aortic arch [13]. The characteristic histopathological features of IgG4-related aortic aneurysm are as follows: i) diffuse fibrous thickening of the adventitia of the aorta (>4 mm), ii) abundant lymphoplasmacytic infiltration with frequent eosinophilic infiltration, iii) abundant IgG4-positive plasma cell infiltration, and iv) the ratio of IgG4-/IgG-positive plasma cells is usually more than 60% [11, 13, 36]. This disease accounts for 5% of all surgically resected abdominal aortic aneurysm, and 57% of inflammatory abdominal aortic aneurysm [11], as well as 7% of thoracic aortic aneurysms [37]. Moreover, IgG4-related aortic aneurysm has been recognized as a manifestation of IgG4-related chronic periaortitis, which encompasses idiopathic retroperitoneal fibrosis, inflammatory aortic aneurysm, and perianeurysmal retroperitoneal fibrosis [38]. Both retroperitoneal fibrosis and IgG4-related inflammatory abdominal aortic aneurysm are recognized as IgG4-related chronic periaortitis, together with mediastinal fibrosis and IgG4-related inflammatory aortic aneurysm of the thoracic aorta [11, 13, 36]. In the present case, although no histopathological analysis of the aorta was performed, the periaortic lesion was suspected clinically to be IgG4-related chronic periaortitis.

In the present case, pericarditis was also observed in the clinical imaging analysis. IgG4-related pericarditis has also been reported as a spectrum of IgG4-related sclerosing disease [39-41], and the pericardium showed the same histopathological and immunohistochemical features as other IgG4-related sclerosing diseases [39, 41]. Moreover, a case with massive pleural and pericardial effusions and thickening of the pericardium has been reported [41]. Therefore, thickening of the pericardium of the present case is suspected to be due to IgG4-related sclerosing disease.

In conclusion, we describe the first documented case of IgG4-related pleuritis and periaortitis. It is important to recognize that IgG4-related sclerosing disease can occur in the pleura, periaortic tissue, and pericardium, and can clinically present as dyspnea and pleural and pericardial effusions. Therefore, detailed histopathological and immunohistochemical analyses and measurement of serum IgG4 are required for correct diagnosis and treatment.

Address correspondence to: Dr. Mitsuaki Ishida, Department of Clinical Laboratory Medicine and Division of Diagnostic Pathology, Shiga University of Medical Science, Tsukinowa-cho, Seta, Otsu, Shiga, 520-2192, Japan. Tel: +81-77-548-2603; Fax: +81-77-548-2407; E-mail: mitsuaki@belle.shiga-med.ac.jp

References

[1] Hamano H, Kawa S, Horiuchi A, Unno H, Furuya N, Akamatsu T, Fukushima M, Nikaido T, Nakayama K, Usuda N, Kiyosawa K. High serum IgG4 concentrations in patients with sclerosing pancreatitis. N Engl J Med 2001; 344: 732-738

[2] Sato Y, Notohara K, Kojima M, Takata K, Masaki Y, Yoshino T. IgG4-related disease: historical overview and pathology of hematological disorders. Pathol Int 2010; 60: 247-258.

[3] Stone JH, Zen Y, Deshpande V. IgG4-related disease. N Engl J Med 2012; 366: 539-551.

[4] Zen Y, Nakanuma Y. IgG4-related disease. A cross-sectional study of 114 cases. Am J Surg Pathol 2010; 34: 1812-1819.

[5] Zen Y, Nakanuma Y. Pathogenesis of IgG4-related disease. Curr Opin Rheumatol 2011; 23: 114-118.

[6] Kamisawa T, Okamoto A. IgG4-related sclerosing disease. World J Gastroenterol 2008; 14: 3948-3955.

[7] Zen Y, Harada K, Sasaki M, Sato Y, Tsuneyama K, Haratake J, Kurumaya H, Katayanagi K, Masuda S, Niwa H, Morimoto H, Miwa A, Uchiyama A, Portmann BC, Nakanuma Y. IgG4-related sclerosing cholangitis with and without hepatic

inflammatory pseudotumor, and sclerosing pancreatitis-associated sclerosing cholangitis: do they belong to a spectrum of sclerosing pancreatitis? Am J Surg Pathol 2004; 28: 1193-1203.
[8] Hamano H, Kawa S, Ochi Y, Unno H, Shiba N, Wajiki M, Nakazawa K, Shimojo H, Kiyosawa K. Hydronephrosis associated with retroperitoneal fibrosis and sclerosing pancreatitis. Lancet 2002; 359: 1403-1404.
[9] Kitagawa S, Zen Y, Harada K, Sasaki M, Sato Y, Minato H, Watanabe K, Kurumaya H, Katayanagi K, Masuda S, Niwa H, Tsuneyama K, Saito K, Haratake J, Takagawa K, Nakanuma Y. Abundant IgG4-positive plasma cell infiltration characterizes chronic sclerosing sialadenitis (Küttner's tumor). Am J Surg Pathol 2005; 29: 783-791.
[10] Yamamoto M, Takahashi H, Ohara M, Suzuki C, Naishiro Y, Yamamoto H, Shinomura Y, Imai K. A new conceptualization for Mikulicz's disease as an IgG4-related plasmacytic disease. Mod Rheumatol 2006; 16: 335-340.
[11] Kasashima S, Zen Y, Kawashima A, Konishi K, Sasaki H, Endo M, Matsumoto Y, Kawakami K, Kasashima F, Moriya M, Kimura K, Ohtake H, Nakanuma Y. Inflammatory abdominal aortic aneurysm: close relationship to IgG4-related periaortitis. Am J Surg Pathol 2008; 32: 197-204.
[12] Kasashima S, Zen Y, Kawashima A, Endo M, Matsumoto Y, Kasashima F. A new clinicopathological entity of IgG4-related inflammatory abdominal aortic aneurysm. J Vasc Surg 2009; 49: 1264-1271.
[13] Ishida M, Hotta M, Kushima R, Asai T, Okabe H. IgG4-related inflammatory aneurysm of the aortic arch. Pathol Int 2009; 59: 269-273.
[14] Kasashima S, Zen Y, Kawashima A, Endo M, Matsumoto Y, Kasashima F, Ohtake H, Nakanuma Y. A clinicopathologic study of immunoglobulin G4-related sclerosing disease of the thoracic aorta. J Vasc Surg 2010; 52: 1587-1595.
[15] Kasashima S, Zen Y. IgG4-related inflammatory abdominal aortic aneurysm. Curr Opin Rheumatol 2011; 23: 18-23.
[16] Zen Y, Kasashima S, Inoue D. Retroperitoneal and aortic manifestations of immunoglobulin G4-related disease. Semin Diagn Pathol 2012; 29: 212-218.
[17] Kawano M, Mizushima I, Yamaguchi Y, Imai N, Nakashima H, Nishi S, Hisano S, Yamanaka N, Yamamoto M, Takahashi H, Umehara H, Saito T, Saeki T. Immunohistochemical characteristics of IgG4-related tubulointerstitial nephritis: Detailed analysis of 20 Japanese cases. Int J Rheumatol 2012; 2012: 609795.
[18] Wong S, Lam WY, Wong WK, Lee KC. Hypophysitis presented as inflammatory pseudotumor in immunoglobulin G4-related systemic disease. Hum Pathol 2007; 38: 1720-1723.
[19] Cheuk W, Yuen HK, Chan JK. Chronic sclerosing dacryoadenitis: Part of the spectrum of IgG4-related sclerosing disease? Am J Surg Pathol 2007; 31: 643-645.
[20] Ishida M, Hotta M, Kushima R, Shibayama M, Shimizu T, Okabe H. Multiple IgG4-related sclerosing lesions in the maxillary sinus, parotid gland and nasal septum. Pathol Int 2009; 59: 670-675.
[21] Taniguchi T, Ko M, Seko S, Nishida O, Inoue F, Kobayashi H, Saiga T, Okamoto M, Fukuse T. Interstitial pneumonia associated with autoimmune pancreatitis. Gut 2004; 53: 770.
[22] Zen Y, Kitagawa S, Minato H, Kurumaya H, Katayanagi K, Masuda S, Niwa H, Fujimura M, Nakanuma Y. IgG4-positive plasma cells in inflammatory pseudotumor (plasma cell granuloma) of the lung. Hum Pathol 2005; 36: 710-717.
[23] Shrestha B, Sekiguchi H, Colby TV, Graziano P, Aubry MC, Smyrk TC, Feldman AL, Cornell LD, Ryu JH, Chari ST, Dueck AC, Yi ES. Distinctive pulmonary histopathology with increased IgG4-positive plasma cells in patients with autoimmune pancreatitis: Report of 6 and 12 cases with similar histopathology. Am J Surg Pathol 2009; 33: 1450-1462.
[24] Zen Y, Inoue D, Kitao A, Onodera M, Abo H, Miyayama S, Gabata T, Matsui O, Nakanuma Y. IgG4-related lung and pleural disease: A clinicopathologic study of 21 cases. Am J Surg Pathol 2009; 33: 1886-1893.
[25] Yamashita K, Haga H, Kobashi Y, Miyagawa-Hayashi A, Yoshizawa A, Manabe T. Lung involvement in IgG4-related lymphoplasmacytic vasculitis and interstitial fibrosis: Report of 3 cases and review of the literature. Am J Surg Pathol 2008; 32: 1620-1626.
[26] Yamamoto H, Suzuki T, Yasuo M, Kobayashi O, Tsushima K, Ito M, Urushihata K, Yamazaki Y, Hanaoka M, Koizumi T, Uehara T, Kawaskami S, Hamano H, Kawa S, Kubo K. IgG4-related pleural disease diagnosed by a re-evaluation of chronic bilateral pleuritis in a patient who experienced occasional acute left bacterial pleuritis. Intern Med 2011; 50: 893-897.
[27] Ishida M, Fukami T, Nitta N, Iwai M, Yoshida K, Kagotani A, Nozaki K, Okabe H. Xanthomatous meningioma: a case report with review of the literature. Int J Clin Exp Pathol 2013; 6: 2242-2246.
[28] Ishida M, Hodohara K, Yoshii M, Okuno H, Nakanishi R, Horinouchi A, Nakanishi R, Harada A, Iwai M, Yoshida K, Kagotani A, Yoshida T, Okabe H. Methotrexate-related Epstein-Barr virus-associated lymphoproliferative disorder occurring in the gingiva of a patient with rheu-

matoid arthritis. Int J Clin Exp Pathol 2013; 6: 2237-2241.
[29] Ishida M, Kodama N, Takemura Y, Iwai M, Yoshida K, Kagotani A, Matsusue Y, Okabe H. Primary bone carcinosarcoma of the fibula with chondrosarcoma and squamous cell carcinoma components. Int J Clin Exp Pathol 2013; 6: 2216-2223.
[30] Ishida M, Igarashi T, Teramoto K, Hanaoka J, Iwai M, Yoshida K, Kagotani A, Tezuka N, Okabe H. Mucinous brobchioloalveolar carcinoma with K-ras mutation arising in type 1 congenital cystic adenomatous malformation: a case report with review of the literature. Int J Clin Exp Pathol 2013; 6: 2597-2602.
[31] Ishida M, Yoshida K, Kagotani A, Iwai M, Yoshii M, Okuno K, Horinouchi A, Nakanishi R, Harada A, Yoshida T, Okuno T, Hodohara K, Okabe H. Anaplastic lymphoma kinase-positive large B-cell lymphoma: A case report with emphasis on the cytological features of the pleural effusion. Int J Clin Exp Pathol 2013; 6: 2631-2635.
[32] Yoshii M, Ishida M, Hodohara K, Okuno Y, Nakanishi R, Yoshida T, Okabe H. Systemic Epstein-Barr virus-positive T-cell lymphoproliferative disease of childhood: Report of a case with review of the literature. Oncol Lett 2012; 4: 381-384.
[33] Miyake K, Moriyama M, Aizawa K, Nagano S, Inoue Y, Sadanaga A, Nakashima H, Nakamura S. Peripheral CD4+ T cells showing a Th2 phenotype in a patient with Mikulicz's disease associated with lymphadenopathy and pleural effusion. Mod Rheumatol 2008; 18: 86-90.
[34] Rossi G, Marchioni A, Guicciardi N, Cadioli A, Cavazza A. Recurrent pleural and pericardium effusions in a white woman with IgG4-related syndrome. Am J Surg Pathol 2009; 33: 802-803.
[35] Sekiguchi H, Horie R, Utz JP, Ryu JH. IgG4-related systemic disease presenting with lung entrapment and constrictive pericarditis. Chest 2012; 142: 781-783.
[36] Ishida M, Okabe H. Immunoglobulin G4-related inflammatory aortic aneurysm. In: Grundmann RT, eds. Etiology, pathogenesis and pathophysiology of aortic aneurysm and aneurysmal rupture. Croatia: InTech, 2011. pp: 91-104.
[37] Kasashima S, Zen Y, Kawashima A, Endo M, Matsumoto Y, Kasashima F, Ohtake H, Nakanuma Y. A clinicopathologic study of immunoglobulin G4-related sclerosing disease of the thoracic aorta. J Vasc Surg 2010; 52: 1587-1595.
[38] Jois RN, Gaffney K, Marshall T, Scott DG. Chronic periaortitis. Rheumatology 2004; 43: 1141-1146.
[39] Sekiguchi H, Horie R, Suri RM, Yi ES, Ryu JH. Constrictive pericarditis caused by immunoglobulin G4-related disease. Circ Heart Fail 2012; 5: e30-31.
[40] Ishizaka N, Sakamoto A, Imai Y, Terasaki F, Nagai R. Multifocal fibrosclerosis and IgG4-related disease involving the cardiovascular system. J Cardiol 2012; 59: 132-138.
[41] Sakamoto A, Nagai R, Saito K, Imai Y, Takahashi M, Hosoya Y, Takeda N, Hirano K, Koike K, Enomoto Y, Kume H, Homma Y, Maeda D, Yamada H, Fukayama M, Hirata Y, Ishizaka N. Idiopathic retroperitoneal fibrosis, inflammatory aortic aneurysm, and inflammatory pericarditis-Retrospective analysis of 11 case histories. J Cardiol 2012; 59: 139-146.