Are There Any Other Organs in Which Autoimmune Pancreatitis-Associated Lesions Remain to be Identified?

Hideaki Hamano¹ and Shigeyuki Kawa²

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Autoimmune pancreatitis has been reported to be associated with various lesions in organs other than the pancreaticobiliary system (1-3). These lesions include salivary gland and retroperitoneal inflammation (4-6), enlargement of the intra-abdominal lymph nodes or hilar and mediastinal lymph nodes (7), interstitial pneumonia (8, 9), pulmonary inflammatory pseudotumor (6, 10), hepatic inflammatory pseudotumor (11-13), and tubulointerstitial nephritis (14, 15). Many of the lesions share two common features: marked improvement with corticosteroid therapy and histopathological findings of prominent infiltration of IgG4-positive plasma cells (1-16).

IgG4-associated prostatitis complicating autoimmune pancreatitis, an informative case for clinicians worldwide reported by Yoshimura et al, indicates that autoimmune pancreatitis may also be associated with lesions in the prostate (17). In 2002, we described patients with autoimmune pancreatitis complicated by retroperitoneal fibrosis who underwent surgery for suspected urinary tract tumors (5). Pathologically, the case showed the common feature of intensive infiltration of IgG4-positive plasma cells into the retroperitonium and pancreas. The case reported by Yoshimura et al provided new evidence that autoimmune pancreatitis may be associated with lesions in the prostate, another organ in the urinary system. Benign prostatic hypertrophy shows age predilection similar to that of autoimmune pancreatitis. However, detailed histopathological examinations, including IgG4 immunostaining, have not been reported in patients with prostatitis or benign prostatic hypertrophy.

A marked difference has been reported between salivary gland lesions complicating autoimmune pancreatitis and those associated with Sjögren’s syndrome; prominent infiltration of IgG4-positive plasma cells are found in the former but not in the latter (3, 18, 19). These findings have recently raised the possibility that the former conditions (Mikulicz’s disease and Kütner’s tumor) may be pathological entities different from Sjögren’s syndrome.

To determine whether this type of prostatic lesion is closely associated with autoimmune pancreatitis, the following two approaches will be needed. One approach is comparison of common prostatitis or benign prostatic hypertrophy with autoimmune pancreatitis-associated prostatic lesions by histopathological examination including IgG4 staining. These conditions may show pathologically significant differences. The other approach should be to investigate whether corticosteroid therapy improves subjective symptoms such as pollakiuria in patients with autoimmune pancreatitis and prostatitis. The results from the two approaches will provide evidence that the prostatic lesions are associated with autoimmune pancreatitis. In the future, when examining a patient with chronic prostatitis of unknown etiology, the urologist may need to consider autoimmune pancreatitis-associated, IgG4-related atypical prostatitis as a differential diagnosis.

Autoimmune pancreatitis may relapse (20, 21). Manifestation of relapse varies, and new lesions may occur in regions other than the pancreaticobiliary system. Most relapses occur after dose reduction of corticosteroids. Some day in the near future, this type of prostatitis may be regarded as a relapse lesion related to autoimmune pancreatitis. As to whether or not there are any other organs in which autoimmune pancreatitis-associated lesions remain to be identified, to date it is unknown.