PERSISTENT PRURITIC SKIN LESIONS WITH SERUM IGG4 LEVELS AT DIAGNOSIS CAN PREDICT HISTOPATHOLOGY AND EXPRESSIONS OF CHEMOKINES, CXCL10, CXCL13, AND CXCXR3, AND AN ENDODENGIN LIGAND S100A8/A9 IN LYMPH NODES OF PATIENTS WITH Adult-onset STILL’s DISEASE

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Background: Adult-onset Still’s disease (AOSD) is a rare systemic inflammatory disease with several symptoms, such as a persistent high spiking fever, typical rash, and lymphadenopathy. Endogenous factors related to interleukin (IL) – 1, such as S100A8/A9 and several chemokines including CXCL10, CXCL13 and CXCXR3, could play a potential role in the pathogenesis of AOSD.

Objectives: We aimed to find out typical histopathologic features, expressed pattern of chemokines in lymph nodes (LN) of AOSD patients.

Methods: Formalin-fixed paraffin-embedded excisional LN tissues from 48 AOSD patients and 6 nonspecific reactive hyperplasia were histologically reviewed. The immunohistochemical stain for CXCL10, CXCL13, CXCXR3 and S100A8/A9 were done. The clinical and laboratory data of the patients who underwent LN biopsies were reviewed.

Results: The LN specimens were categorised according to four distinctive patterns: follicular (n=2, 4.2%), paracortical (n=19, 39.6%), diffuse (n=9, 18.8%) hyperplasia, or mixed pattern (n=18, 37.5%). The other examined histologic features were presence of necrosis, karyorrhexis, immunoblastic, histiocytic and vascular proliferation. Most of the cases were required to take into differential diagnosis such as dermatopathic lymphadenitis (n=16, 33.3%), lymphoma (n=11, 22.9%) and histiocytic necrotizing lymphadenitis (n=9, 18.8%). The expression of chemokines and S100A8/A9 were higher than that of nonspecific reactive hyperplasia. The expression of chemokines and S100A8/A9 were more expressed in AOSD patients than those of reactive hyperplasia, they may serve as a pathogenesis of AOSD.

Conclusions: Histopathologic findings of LN in AOSD patients are diverse enough to be included various differential diagnosis. Because the several chemokines and S100A8/A9 were more expressed in AOSD patients than those of reactive hyperplasia, they may serve as a pathogenesis of AOSD.

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