Diagnosis: Multicentric plasma cell type Castleman's disease

Histopathological revision of lymph nodes revealed lymph node structure with mild architectural alternation and obliteration of some of the subcapsular sinusoids due to extensive increase of lymphoid follicles both in cortical and medullary areas. In addition to above morphologic findings, the interfollicular areas were loaded with sheets of plasma cells. On immunohistochemistry staining, CD20 highlighted the localization of B cells in germinal centers. The CD31 also highlighted the rich vascularization of the lymph node. On serial sections, there was no evidence of any CD30 positive atypical lymphocytes.

The above morphological findings were not characteristic and could be seen in a variety of diseases, like lymphoma, collagen vascular disorders, infections (especially HIV) (1) and Castleman's disease.

Physical examination and serologic tests for rheumatologic diseases, HIV and other suspected etiologies like EBV, CMV and HHV-8 were negative. For further evaluation we examined lymph node biopsy with PCR to identify HHV-8. The positive HHV-8 PCR of lymph node sampling, with exclusion of other differential diagnoses and suggestive pathologic features resulted in diagnosis of Castleman's disease.

Castleman’s disease (CD) was introduced in 1956 as a localized mediastinal lymph node hyperplasia resembling thymoma (2). Since 1956, multiple cases of CD have been reported and new data about its presentations, localization, etiology and treatment have been collected. However, it is a rare entity and a mimicking disease. At present, two distinct pathologic types (hyaline vascular pattern and plasma cell type) and also two distinct clinical manifestations (localized versus multicentric) are well known (3). Before 90s, HHV-8 had been named Kaposi's sarcoma associated herpes virus (KSHV) (4), but after identification of its role in pathogenesis of other conditions we also consider HHV-8 as one of multiple etiologies of Castleman's disease (4-6).

Identification of HHV-8 in tissue in this case is the first report in this respect from Iran.

Recently, valganciclovir has been used in a randomized clinical trial for reducing HHV-8 shedding. Limiting HHV-8 replication appears to control some cases of Castleman's disease (7). We initiated prednisolone as the first line regimen, but considering the persistent mediastinal lymphadenopathy and since the symptoms remained unchanged after 45 days, she is supposed to take valganciclovir and will probably be treated by chemotherapeutic agents (8).

REFERENCES


