

Comparison between IgG and IgM type anti-alpha-enolase antibody in patients with Behcet's disease

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Behcet's disease (BD) is a chronic inflammatory disease of unknown etiology, characterized by recurrent oral and genital ulcers, skin lesions, uveitis, and arthritis. It is regarded as vasculitis and anti-endothelial cell antibodies are found in BD. One of the endothelial cell antibodies was reported to recognize alpha-enolase and serum IgM anti-alpha-enolase antibody (AEA) was associated with involvement of the vascular system in BD in previous study. This study was aimed to investigate expression of alpha-enolase in the surface of peripheral blood cells and serum IgM or IgG AEA, and their association with clinical manifestations or disease activity of BD. Cell surface alpha-enolase expression was examined from several cell types of peripheral blood, including lymphocytes, monocytes, and granulocytes using flow cytometry in patients with BD and healthy controls (HCs). Serum AEA levels were measured by enzyme-linked immunosorbent assay (ELISA) in 110 BD patients and age/sex matched 110 HCs. Association of alpha-enolase or AEA with clinical manifestation was analyzed. The frequency of surface alpha-enolase-expressing cells was increased in BD in lymphocytes and monocytes. Serum IgG AEA levels were increased in BD patients (median [IQR], 0.360 [0.268-0.482], $p < 0.0001$), particularly in active patients (BDCAF ≥ 2) (0.375 [0.274-0.537], $p < 0.0001$) compared to HCs (0.274 [0.231-0.357]). The levels of IgG AEA were correlated with the number of oral ulcer, ESR, and CRP. There was no difference of IgM AEA in sera from BD compared to HCs (0.538 [0.351-0.763] vs. 0.504 [0.344-0.823], $p = 0.764$) and no association of AEA levels and clinical manifestations in patients with BD. In conclusion, serum IgG AEA was increased in BD patients and correlated with oral ulcer, ESR and CRP.

A case of atlantoaxial subluxation caused by odontoid osteoradionecrosis

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Introduction: Atlantoaxial subluxation (AAS) is a condition characterized by dislocation between the atlas (C1) and axis (C2), which often manifests in rheumatoid arthritis (RA) or spondyloarthropathy (SpA). Here, we report a case of AAS, which was later found to be a delayed complication of radiotherapy. **Case:** A 57-year-old man was referred to the rheumatology clinic with right occipital neck pain combined with arm radiculopathy and finding of AAS on MRI. He had been diagnosed with nasopharyngeal cancer 7 years previously, and was treated with concurrent chemoradiotherapy using cisplatin and 75.6 Gy of radiotherapy. On physical examination, there was no evidence of peripheral arthritis or psoriasis. There was no loss of lower lumbar motility or tenderness in the gluteal region. In laboratory tests, inflammatory markers were elevated (erythrocyte sedimentation rate 70 mm/h and C-reactive protein 2.76 mg/dL), but rheumatoid factor, anti-citrullinated protein antibody and HLA-B27 were negative. Sacroiliac joint X-ray was normal. CT of the cervical spine revealed widening of the atlanto-odontoid joint and severe moth-eaten osteolytic lesion in the odontoid process. Due to the patient's history, PET-CT was performed and the C1-2 spine showed increased FDG uptakes suggesting bone metastasis. After considering all treatment options, C1-3 posterior fusion was performed with biopsy of adjacent bone and ligament. The biopsy revealed chronic inflammatory changes with fibrosis in the absence of malignant cells, which was compatible with osteoradionecrosis. These observations suggested that the AAS was induced by radiotherapy for nasopharyngeal cancer. After surgery, the patient's symptoms subsided and he has been followed up for more than 1 year without neurological symptoms. **Conclusions:** AAS is usually encountered in RA and SpA. In this case, various causes of AAS were ruled out starting from rheumatological disease to malignancy, and a final diagnosis of osteoradionecrosis was made. Odontoid osteoradionecrosis is rare but should be considered in differential diagnosis of the cause of AAS in patients who have previously received radiotherapy for head and neck cancer.