Takayasu's arthritis associated with chronic hepatitis B

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We report a case with Takayasu's arthritis, which is a unique autoimmune disease of unknown etiology co-existing with chronic hepatitis B.

A 26-year old female patient was suffering from diffuse myalgia and arthralgia. During her physical examination blood pressures of 100/70 mmHg on her left and 80/40 mmHg on her right arm were measured ; no palpable pulses over the brachial, ulnar and radial arteries were present. Bruises were noted over both carotid arteries and over the left side of the abdomen. Laboratory tests showed an increased erythrocyte sedimentation rate (ESR) of 120 mm/h and C - reactive protein of 20 mg/L. The diagnosis of Takayasu's arthritis was confirmed by angiography. In the angiography subtotal stenosis of the right carotid artery and a 50% stenosis of the left carotid artery were found. (Corticosteroid therapy of) metylprednisolon 8 mg/day was started. After administering the corticosteroid medication, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were elevated at levels of 462 IU/l and 324 IU/l respectively. Positive hepatitis B surface antigen (HBsAg), negative hepatitis B early antigen (HBeAg) and hepatitis B core antibody (HBcIgM) were found. 40000 copies/ml HBVDNA was detected. The liver biopsy was graded by Knodell's Histological activity index (HAI) (1) and revealed chronic hepatitis B necroinflammatory activity of 9 and fibroblastic activity of 2. Lamivudine treatment was started with a dosage of 100 mg/day. After six months of treatment HBVDNA was no longer detectable. The patient's vasculitis continued ; she had arthralgia with an increased ESR of 70 mm/h and C-reactive protein of 15 mg/L.

Hepatitis B surface antigen containing immune complexes as the cause of the vascular injury in systemic necrotizing vasculitis such as polyarthritis nodosa is well-established (2). But the association between Takayasu's arthritis and hepatitis has rarely been described. Thorleif et al. reported a case of Takayasu's arthritis with insular infarction and chronic hepatitis B (3). Another report described a family of four sisters being HBsAg positive in which one daughter developed Takayasu's arthritis (4). There are two more reports about Takayasu's arthritis after inoculation with plasma-derived hepatitis B vaccine (5,6).

(To our knowledge) The etiology of Takayasu's arthritis is unknown but it could be related to infection, endocrine abnormalities and autoimmune mechanisms (7). Activation of the immune system by a viral disease could be associated with the onset or progression of Takayasu's arthritis. In our patient after one year of follow-up, HBVDNA was still negative but HBsAg was positive. The hepatitis B surface antigen could have triggered an immunological inflammatory process responsible for the arteritis in our patient. The reported cases about Takayasu's arthritis after inoculation with plasma-derived hepatitis B vaccine lead us to consider possible relationship between HBsAg and vasculitis (5,6).

Lamivudine is well-tolerated oral nucleoside analoge and available for the treatment of chronic hepatitis B (8). Antiviral drugs decrease viral load and promote HBsAg seroconversion (9). In our patient, after one year of lamivudine therapy there was sustained suppression HBVDNA replication. Lamivudine treatment did not change Takayasu's disease activity as reflected by a high serum sedimentation rate.

In conclusion, the findings in our patient suggest that the hepatitis B surface antigen induced cellular immune response might lead to Takayasu's arthritis. However, coincidence could not be excluded. Lamivudine treatment did not affect the disease activity but suppressed HBVDNA. While evaluating Takayasu's arteritis further investigations should be done about pathogenesis and especially viral antigens must be taken into consideration.

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