Takayasu’s arteritis with transient clubbed finger

Sirs,

Takayasu’s arteritis is a disease in which chronic inflammation affects the aorta and its branches (1). Ishikawa et al. (2) reported that vascular stenosis most commonly affects the left subclavian artery and then the right subclavian artery in this disease. Finger clubbing in Takayasu’s arteritis has been reported by Kaditis et al. (3) in only one patient, who had unilateral clubbing. The diseases linked with finger clubbing include lung cancer, lung abscess, vascular shunting, liver cirrhosis, and inflammatory bowel disease (1). We had a patient with Takayasu’s arteritis who developed transient clubbing early during the course of her disease.

The patient was a 27-year-old woman who presented with coldness of the fingers of both hands. The patient had suffered from dullness in the fingers of the right hand since 10 months before admission. She presented to a local hospital 7 months before admission. There was a difference in blood pressure between the two arms. She was referred to a cardiovascular hospital and underwent IVDSA. 90% stenosis of the right subclavian artery was detected on the angiogram. The patient developed a fever and clubbing of the fingers of the right hand 3 months before admission, and clubbing of the fingers on the left side also occurred subsequently.

The patient was referred to our hospital in October 1994. On admission, she had a body temperature of 37.3°C. Her blood pressure was 110/70 mmHg in the right arm and 90 mmHg in the left arm. A bruit was heard in the bilateral carotid arteries. Clubbed fingers were noted bilaterally with especially prominent changes in the right hands (Fig. 1). Hematologic tests indicated mild anemia, with hemoglobin 10.7 g/dl. The WBC was 7500/µl. Her erythrocyte sedimentation rate (ESR) was 73 mm/hr. Blood chemistry tests revealed no abnormalities in hepatic enzymes, renal function, or electrolytes. CRP was increased to 5.4 mg/dl, and the serum IgG level was slightly increased at 2150 mg/dl. One month after admission, IVDSA revealed marked stenosis of the right axillary artery with a collateral pathway. The left axillary artery was completely obstructed, and a collateral pathway had formed.

After admission, prednisolone was started at a dose of 40 mg/day, decreasing the ESR from 73 mm/hr to 6 mm/hr and the CRP level from 5.4 mg/dl to 0.58 mg/dl. The finger clubbing also showed improvement. However, the weak right radial artery pulse was not improved. The finger clubbing resolved almost completely 3 year and 1 month after admission (Fig. 1).

In this patient, finger clubbing developed at an early stage of Takayasu’s arteritis. The association of finger clubbing and Takayasu’s arteritis has already been reported by Kaditis et al. (3), who observed unilateral clubbing in a patient with this disease. The proposed mechanisms of the development of clubbing in these diseases include peripheral tissue hypoxemia, and increased peripheral tissue blood flow mediated through a neural reflex (4). Large fragments of megakaryocytes released from the bone marrow normally enter the pulmonary circulation and are fragmented in the pulmonary vascular bed (1). When an A-V shunt is present, such fragments do not enter the pulmonary circulation, instead directly entering the systemic circulation and thus reaching the capillaries. Platelet derived growth factor (PDGF) is released from megakaryocyte fragments and collagen fibers start to proliferate as a result of increased capillary permeability.

In our patient, clubbing developed in the right hand following the onset of ischemia related to right subclavian artery stenosis. Stenosis then also affected the left subclavian artery, leading to finger clubbing in the left hand. These symptoms were improved by steroid therapy. The occurrence of clubbing in association with Takayasu’s arteritis has only been reported by Kaditis (3) in one patient, suggesting that finger clubbing occurred due to this subclavian stenosis. In our patient, it is thought that clubbing occurred due to subclavian artery stenosis and the subsequent circulatory disturbance in the peripheral tissues. Since clubbing occurred ipsilaterally with the progression of subclavian artery stenosis and resolved after the commencement of steroid therapy, together with improvement of the vascular symptoms, it is suggested that the finger clubbing occurred in association with stenosis resulting from vascular inflammation.

Although clubbing is not common in the presence of subclavian artery stenosis, it probably occurred in our patient because of severe vascular inflammation with the rapid progression of stenosis. The ischemic symptoms and clubbing were alleviated by steroid therapy. The clubbing may have been improved as a result of increased blood flow, but it is also possible that the symptoms were improved by inhibition of the release of cytokines such as PDGF by steroid therapy. Although clubbing has only rarely been reported in patients with Takayasu’s arteritis, its occurrence in the early stage of this disease may have etiologic implications.

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Letters to the Editor

References

Synovial immunoreactive beta-endorphin levels in rheumatoid arthritis and osteoarthritis

Sir,
The role of beta-endorphin (BE) in the pathogenesis of inflammation and the mechanism of its analgesic action are exciting subjects, neither of which have been fully elucidated as yet. Recently Conti et al. reported that BE levels are decreased in chronic fatigue syndrome (1). There are only a few studies in the literature focusing on the presence of BE levels in synovial fluid or synovial tissue and mainly in the cerebrospinal fluid (CSF). Denko et al. found higher BE levels in the synovium of rheumatoid arthritis (RA) patients compared to osteoarthritis (OA) patients (2). The presence and analgesic action of opioid peptides in the inflamed tissues have been demonstrated by Stein et al. (3). In RA, according to Suzuki’s studies on synovial fluid and Yoshino’s studies on synovial culture supernatant, BE levels are elevated in both patients (r = 0.22) than in OA patients (r = 0.03). We subsequently measured the BE level in the CSF of 9 other patients suffering from hip osteoarthritis, who were undergoing total hip arthroplasty. The mean immunoreactive BE level was 7.27 ± 1.98 fmol/l. The CSF BE level was measured only in OA patients.
The hypothalamic-pituitary-adrenal axis has been shown to be deficient in RA (11). Stress is considered to play a role in RA and pain is a cardinal manifestation of this disease. The synovial BE level in our study was slightly higher in OA patients than in RA patients. In agreement with the data in the literature, we found a negative correlation between the BE level and ESR values (7). Although the ratio of synovial, serum, and CSF levels may vary depending on the anti-serum used to perform the assay, these (and our previous) results suggest that the synovial BE level is much higher compared to the serum and CSF levels. The mechanism of the analgesic action of BE is extremely complicated. BE levels are presumed to be reduced in the cerebrospinal fluid of patients with chronic pain syndromes. Direct monitoring of BE levels in the cerebrospinal fluid is extremely difficult in humans; however, the evaluation of changes in peripheral tissues could yield valuable information. Opioid peptides are present in inflamed synovial tissues. Besides the gate control theory of pain, the peripheral action of BE plays an important role (inhibiting nociception) in decreasing the pain in the peripheral tissue. In conclusion, the role of BE in the pathogenesis of inflammation as well as the mechanism of its analgesic action are exciting research topics worth further investigation.

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References

Table I. Synovial fluid beta endorphin (BE) and ESR levels in RA and OA.

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<th>RA (n = 9)</th>
<th>OA (n = 9)</th>
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<tr>
<td>BE (fmoI/l)</td>
<td>75.2 ± 20.8</td>
<td>96.8 ± 41.79</td>
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<td>ESR (mm/h)</td>
<td>62 ± 22.6</td>
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