■ S-739 ■

A case of severe gouty tophi-induced carpal tunnel syndrome: Operative finding and its outcome

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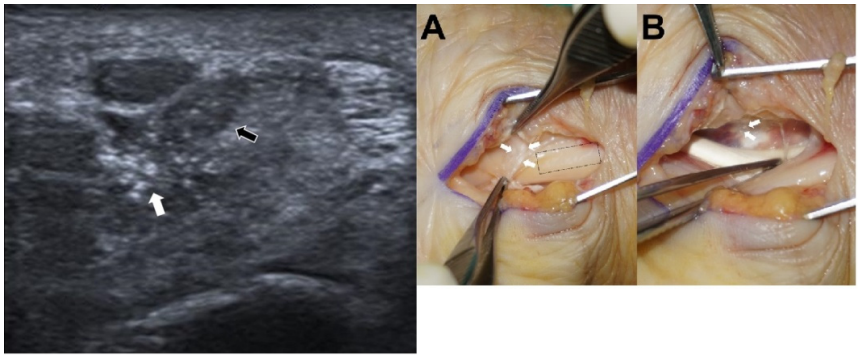
Carpal tunnel syndrome (CTS) refers to compressive neuropathy of the median nerve in the wrist and is the most common neuropathy of the upper extremity. Although CTS concomitant with gout has been addressed in several reports , CTS caused through tophi deposits situated directly over the median nerve has been rarely reported in the literature A 75-year-old woman had swelling and tenderness in both hands, wrists, ankles, and first metatarsophalangeal (MTP) joints. She had a nerve conduction velocity (NCV) test for sensory nerve conduction in the right median nerve (finger-wrist) showed that latency, amplitude, and conduction velocity were 4.5 ms, 4.4 uV, and 28.8 m/s, . We did operation for her CTS. We identified that multiple tophi were deposited in the synovial membrane and sheaths. We did multiple synovectomy, with tophi extraction from the surrounding flexor tendon and the median nerve, and performed a median nerve neurolysis. After surgery, the nocturnal paresthesia and burning pain disappeared as did the persistent fingertip numbness and motor weakness bilaterally. At the 4-week follow-up, the NCV test for sensory nerve conduction indicated latency, amplitude, and conduction velocity were 4.4 ms, 12.45 uV, and 29.5 m/s, respectively. In conclusion, we observed a rare case of gout tophi deposition in synovium and sheaths on the median nerve, which caused CTS symptoms. In addition, surgical treatment can be helpful for treating CTS due to gout-related arthritis.



■ S-740 ■

Association of mitochondrial DNA copy number with disease activity in rheumatoid arthritis

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Background: Mitochondrial biology are associated with inflammation. Alteration of mitochondrial DNA copy number, which reflects oxidant-induced cell damage, has been observed in a wide range of human diseases. Here we examined the comparison of the mitochondrial DNA copy number between rheumatoid arthritis (RA) patients and healthy controls (HC). **Method:** 41 RA patients and 45 age- and sex-matched healthy controls were recruited. There were 15 patients of clinical remission group with DAS28-ESR≤2.6 and 20 patients of non-clinical remission group with DAS28-ESR＞2.6. The mitochondrial DNA copy number was measured by a quantitative real-time PCR assay using DNA extracted from peripheral blood. **Results:** The analyses show significant differences in mitochondrial DNA copy number between RA and HC (mean of RA＝101.71, interquartile range (IQR): 71.84-154.08; mean of HC＝140.56, IQR: 103.43-195.07; *p* value＝0.012). By disease activity, however, mitochondrial DNA copy number of clinical remission group was not different from non-clinical remission group (*p* value＝0.268). **Conclusion:** Our results suggest that the more mitochondrial DNA copy number is one of the characteristics of rheumatoid arthritis compared with healthy controls.