Visualization of extensor digitorum tenosynovitis with three-dimensional ultrasonography

Sir, Inflammation of tenosynovium is a kind of synovitis characterized by synovial thickening and collected effusion around the tendons [1]. Musculoskeletal ultrasonography gives us a new way of assessing the inflammatory status of synovium by measuring thickness of synovium and depth of retained fluid. However, conventional 2D ultrasonography has some limitations [2]. First, irregular synovial lesions such as focal nodular hypertrophy are sometimes not evaluated adequately with standard-view ultrasonography, which just checks the cross-section of synovium. Second, it is not easy to understand the surface morphology of target organ with conventional 2D ultrasonography, because 3D reconstruction with imagination requires a lot of examiner experience. To compensate for these shortages, we decided to extend conventional 2D ultrasonography into a 3D method the technique that has been widely available in obstetric clinics for examining and following up the fetus [3]. This technique is useful because it is safe as a diagnostic tool and such a sensitive diagnostic method that even a small anomaly of fetal surface can be easily sought. Moreover, more perceptive imaging can be made by 3D ultrasonography. Simple scanning on the abdomen of pregnant woman makes it possible to get 3D reconstructed image of fetus with computerized process automatically (Fig. 1A). We applied this method to evaluate the rheumatic diseases whose surroundings were similar to that of uterus containing a fetus. It was useful to examine the organs that contained fluid or were surrounded by fluid. Here, we show the case of a woman with tenosynovitis of extensor digitorum longus at wrist, which was surrounded by fluid.

A 45-yr-old woman visited our hospital because of painful swelling in her right wrist and polyarthralgia of hands with long-lasting morning stiffness. Rheumatoid factor was positive and
anti-CCP antibody was also positive. She had an erythrocyte sedimentation rate (ESR) of 90 mm/h and C-reactive protein (CRP) of 7.8 mg/dl. Radiology showed several erosions at the wrist and metacarpal joints. She began to be treated with the diagnosis of rheumatoid arthritis. Prednisolone (7.5 mg/day), non-steroidal anti-inflammatory drug and methotrexate (10 mg/week) led to the relief of morning stiffness and polyarthritis. Ultrasonography of the swollen wrist showed the synovial proliferation and fluid accumulation around the extensor digitorum tendon at the level of the wrist, suggesting the diagnosis of tenosynovitis of extensor digitorum tendon (Fig. 1B). Then cross-sectional 2D images were gathered at two areas, distal and proximal lesion (Fig. 1C). 3D ultrasonographic scanning was performed on this tenosynovitis of extensor digitorum tendon. 3D reconstruction of tenosynovitis showed more detailed and perceptive information about tendon surface and fluid amount (Fig. 1D).

To the best of our knowledge, it is the first attempt to apply 3D technology to visualize tenosynovitis. The clinical impact of 3D ultrasonography was uncertain. However, the scanning method used in 3D ultrasonography is much simpler than 2D, because probe of 3D is just pressed against the lesion without swiping or tilting. Simpler method means the potential of 3D method to reduce inter-personal variation in future, which has been pointed out as a major weakness of ultrasonography.

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Development of active tuberculosis following initiation of infliximab despite appropriate prophylaxis

Sir, Prospective cohort studies have demonstrated an increased risk of active tuberculosis in patients treated with tumor necrosis factor antagonists (anti-TNF agents) [1]. Current guidelines support chemoprophylaxis with isoniazid (INH) for 6-9 months prior to the initiation of anti-TNF agents in patients with evidence of latent tuberculosis [2]. We present two rheumatoid arthritis patients who had positive tuberculin skin tests (TST). Despite receiving an adequate course of INH for latent tuberculosis infection, both patients developed active tuberculosis after initiation of infliximab.

Patient 1 is a 58-yr-old male Chinese immigrant with a 3yr history of seropositive rheumatoid arthritis who was admitted to the hospital with three weeks of fevers, chills, productive cough, night sweats and altered mental status.

His medical history was otherwise notable for chronic inactive hepatitis B and chronic obstructive pulmonary disease. Three yrs prior, he had a positive TST. Chest computed tomography (CT) revealed emphysematous changes and multiple calcified granulomas consistent with prior tuberculosis. He was treated with a 9 month course of INH, 300 mg/day.

Despite treatment with methotrexate and sulfasalazine, his rheumatoid arthritis remained active, requiring frequent intra-articular steroid injections and oral corticosteroids. Fourteen months prior to admission he started infliximab infusions (6 mg/kg) in addition to methotrexate and sulfasalazine, and was successfully tapered off corticosteroids.

On admission, the patient was ill appearing and thin. He had a temperature of 102.6° with normal respiratory rate and oxygen saturation. The breath sounds were decreased and there were diffuse expiratory wheezes in all the lung fields. There was no lymphadenopathy.

Initial investigations included normal complete blood counts, electrolytes, liver and renal function tests, as well as negative tests for HIV, hepatitis C and rapid plasma reagent. Sputum stains for acid-fast bacilli, pneumocystis carinii and legionella species were negative.

A chest radiograph and chest CT scan demonstrated no new infiltrates or pathological lymphadenopathy. A CT scan of the abdomen and pelvis revealed enlarged mesenteric and retroperitoneal nodes. Bone marrow biopsy and right inguinal lymph node biopsy did not show evidence of malignancy or granulomatous disease.

Given the high level of concern for tuberculosis, the patient was empirically started on INH, rifampin, pyrizinamide and ethambutol. Nine days after discharge the sputum cultures grew mycobacterium tuberculosis, with sensitivity to ethambutol, INH, pyrazinamide, rifampin and streptomycin. He has since made a full recovery.

Patient 2 is a 77-yr-old woman originally from Trinidad with seronegative rheumatoid arthritis treated with infliximab who was admitted to the hospital with a three week history of worsening weakness and fatigue. One week prior to admission she developed a non-productive cough, fevers and chills. On the day of admission she was profoundly weak and was noted by family members to have slurred speech.

Her medical history was also notable for sickle cell trait, glucose-6-phosphate dehydrogenase deficiency, sideroblastic anaemia and a single episode of bilateral pulmonary embolism. Ten yrs prior she had a positive TST; chest radiographs did not show any abnormalities suggestive of tuberculosis at the time. Seven yrs prior to admission she was treated with a 7 month course of INH after starting prednisone. She had a negative TST 4 yrs prior. She had previously failed methotrexate and subsequently failed combination therapy with leflunomide. She was started on treatment with infliximab at 4 mg/kg every 6 weeks 4 months earlier. Medications on admission included methotrexate (17.5 mg orally once per week), prednisone (5 mg/day), infliximab, warfarin and oxycodone.

On admission, her temperature was 103 F and oxygen saturation was 92%. On chest auscultation, there were inspiratory crackles at the left lower lung base. She had right hip pain with very limited range of motion. Initial laboratory evaluation was notable for a white blood cell count of 2600 cells, with 59% neutrophils and 9% bands. Serum electrolytes were normal; blood...