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Delayed Onset Asymmetrical Swan-Neck Deformity in a Post-Stroke Patient : A Case Report

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Introduction

Swan neck deformity(SND) is characterized by hyperextension of the proximal interphalangeal(PIP) joint and flexion of the metacarpophalangeal(MCP) and distal interphalangeal joint(DIP). Though it is frequently reported to be caused by rheumatoid arthritis, four major categories of clinical conditions including rheumatologic diseases, neurologic problems, hypermobility syndromes and traumatic causes were reported to be associated with SND. We present a case of the post-stroke patient mainly presenting with left side weakness showing delayed onset SND on his right hand.

Case report

A-45-year old right-handed male was admitted to the department of rehabilitation medicine at our hospital for comprehensive rehabilitation therapy. 9 months ago, He suffered from left basal ganglia and intraventricular hemorrhage.(Fig.1.) There was hyperextension of PIP joint and flexion of DIP joint on his 3rd, 4th finger showing a swan-neck deformity with his left hand spared (Fig.2.). There was no previous history of rheumatologic diseases and no evidence of traumatic injury on right hand. At the time of admission, he was bed-ridden state and relied on wheelchair for functional mobility with the K-MMSE of 14. Muscle strength in the right and left upper extremity was graded as three and zero, respectively. The spasticity of the both upper extremity was difficult to evaluate accurately due to poor cooperation. On physical examination, there was no evidence of swelling or erythema on finger joint. But, the patient present pain while we passively flex and extend the finger joint. Additionally, burnell-littler test showed positive suggesting intrinsic muscle tightness. The laboratory test revealed normal CRP, ESR, negative rheumatoid factor and anti-CCP antibody. Radiographic finding of both hands was normal except periarticular osteopenia. Ultrasonography of the finger showed no evidence of synovitis of MCP, PIP joints, and a thickening of the dorsal sided PIP joint capsule and tendinosis of central hood of extensor tendons of the right 3rd and 4th fingers were noted. As his symptoms might be attributed to intrinsic muscle spasticity causing stretching of the PIP volar plate over time, MRI of the brain was done. A newly appeared enhanced lesion in left internal capsule in axial T2-weighted MRI images was found, 9 months after the onset.(Fig.3.)

Discussion

At first, we thought that musculoskeletal disorders might be attributed to the development of ipsilateral SND in our post-stroke patient. However, the opposite side of the brain lesion was proved to be the cause. Although not common, neurologic disorders such as cerebrovascular accident can develop SND. It seems that intrinsic muscle

hyperactivity seems to be predominant mechanism. For the clinicians, it is important to know the stroke as the possible cause of this deformity so that early diagnosis andtimely intervention that leads to better functional outcome can be applied.

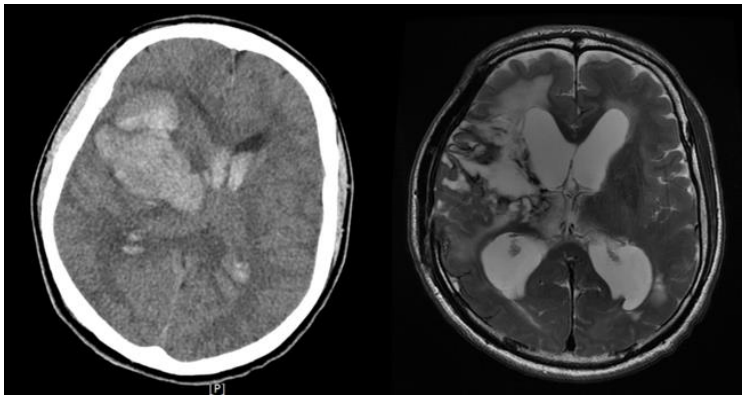

fig1. CT scan of brain demonstrating large intracerebral and intraventricular hemorrhage and T2-weighted fast spine echo MRI of brain taken 3 weeks after the onset.

fig2. Asymmetrical swan-neck deformity of the right hand

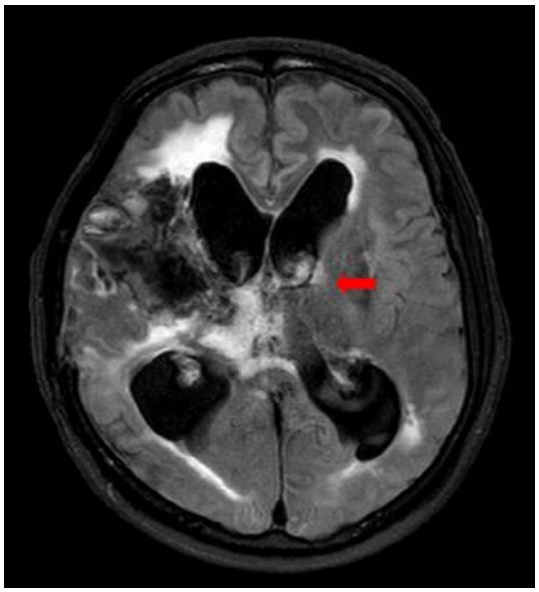

fig3. T2-weighted FLAIR axial MRI taken 9 months after the onset shows ventriculomegaly, tissue loss and encephalomalacia in the right deep gray matters as well as infarction in left BG(arrow)