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Scientific Research and Essays

Case Report

Interesting finding of hyperpyrexia relieving post-stroke spasticity: A case report

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As an important cause of disability in adults, post-stroke spasticity (PSS) is a big challenge for the clinician and the effective management of spasticity should be necessary and essential for patients. We herein present a rare case of hyperpyrexia caused by upper respiratory tract infection which obviously relieved post-stroke spasticity (PPS), inspired by this, hyperthermia might be a new good way to relieve spasticity.

Key words: Post-stroke, hyperpyrexia, spasticity.

INTRODUCTION

post-stroke spasticity (PSS) is emerging as an urgent issue for stroke survivors, and is greatly associated with severe impairments, dis-function, and low health-related quality of life. Consequently, more and more rehabilitation resources are used in relieving the condition (Cousins et al., 2009). Treatment interventions for post-stroke hypertonicity include stretching, splinting, strengthening of antagonist muscles, oral medications, focal injections (phenol or botulinum toxins) and, less commonly, surgery (Marciniak, 2011). Following, the interest that we will describe, perhaps will make us transform the idea of relieving spasticity, we found that hyperpyrexia can relieve post-stroke spasticity in this case.

CASE REPORT

A 51-year-old woman was admitted to the emergency room in our hospital because of a sudden onset of headache and left hemiplegia. Immediate, CT scan showed an intra-cranial hemorrhage on the right side of the basal ganglia (Figures 1 and 2). After six hours, she accepted the surgery, and the surgery went well. Two weeks later, to further improve the physical activity, she was transferred to rehabilitation ward. Baseline rehabilitation evaluation and assessment revealed: Broca aphasia, hemidysesthesia, motor function: Fugl-Meyer assessment (10 points), Brunnstrom assessment (left upper/lower extremity and left hand:scale I), and the muscle tone of the left side: hypotonia. Activities of daily living were evaluated with Barthel index: 10 point. She received systematic rehabilitation treatment, after 3 months, muscle tone increased, modified ashworth scale showed that: left upper /lower extremity: scale II, left hand I^{+}) many measures were taken to relieve spasticity: continuous passive traction, Myonal (the maximum application was 300 mg per day)/ botulinumtoxin type A(100 μ local injected in left biceps) and local hyperthermia

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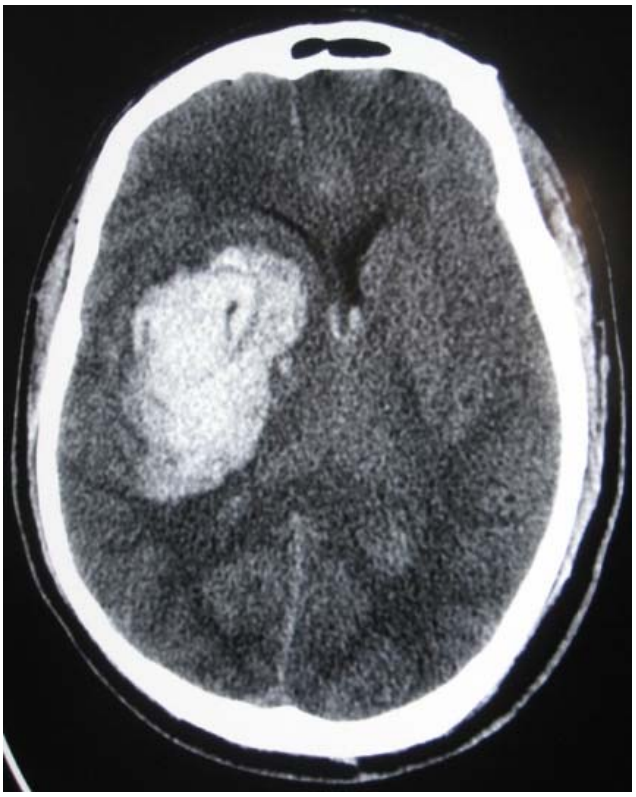

Figure 1. Preoperative CT scan. The intra-cranial hemorrhage in the right side of the basal ganglia.

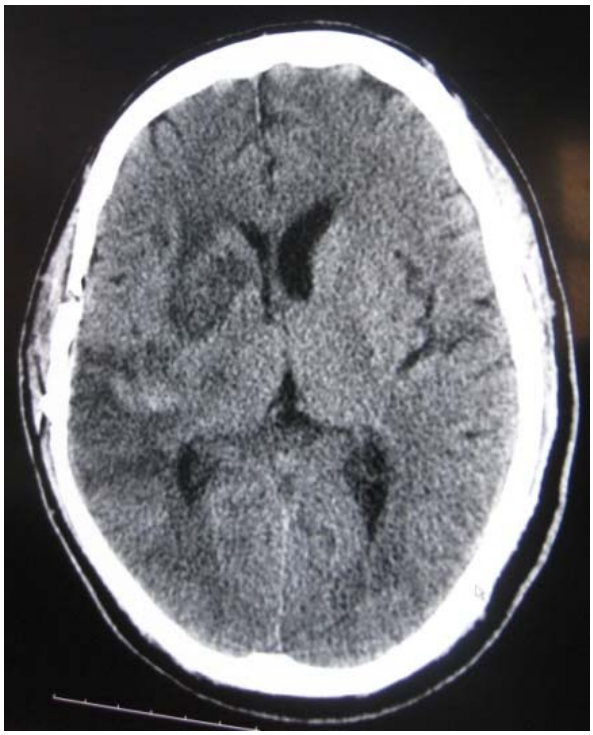

Figure 2. The CT findings after the right basal ganglia hematoma removal surgery.

therapy (Wax therapy on the left upper extremity) to relieve hypertonia. However, the effects were temporary and not so obvious. 15 months after brain hemorrhage, the patient suffered from upper respiratory tract infection, and developed flu symptoms with a temperature of 39.4°C, with malaise, headache and anorexia. 5 min later, she was given indometacin 50 mg externally. After 1 h, the symptoms gradually relieved her, and there is no use of other drugs. The next day, surprisingly and interestingly, Hemiplegic limb spasticity was obviously relieved. Rehabilitation evaluation revealed: Broca aphasia, motor function: Fugl-Meyer assessment (47 points), Brunnstrom assessment (left upper/lower extremity:scale III+; left hand:scale IV), and the muscle tone of the left side: Modified ashworth scale showed that: left upper /lower extremity: scale I^{+} left hand I. Activities of daily living was evaluated with Barthel index: 55 point. Hemiplegic limb spasticity was obviously relieved. This situation sustained for about 1 month.

DISCUSSION

Spasticity is defined as “disordered sensorimotor control, resulting from an upper motor neuron lesion, and presenting as intermittent or sustained involuntary activation of muscles” (Pandyan et al., 2005). The mechanisms of spasticity were not completely understood, post-stroke spasticity (PSS) is a polysynaptic muscle reflex caused by abnormal processing of proprioception in the spinal cord (Sherman et al., 2000), also it is thought to be related to change in the balance of excitatory and inhibitory inputs to the motor neuron pool (Ozcakir and Sivrioglu, 2007). General consensus is that PSS seriously impacts post-stroke victims. Motor ability and activity performance is closely relative with the PSS, some of the secondary complications, such as contracture, pain, and weakness also caused by PSS, which might further contribute to post-stroke disability (Sommerfeld et al., 2004). However, heterogeneity across studies and epidemiological data regarding PSS were limited, it was difficult to qualify the prevalence and impact of PSS. Data obtained from some clinical studies, the prevalence of PSS ranged from 4 to 42.6%, and the rate in the chronic phase of stroke (>3 months) was highest (Brainin et al., 2011).

In clinical practice, a large variety of treatment options can be used to relieve post-stroke spasticity, and multidisciplinary interventions after stroke often lead to best clinical outcomes. The measures adopted in the former patient include continuous passive traction, baclofen, Botulinum toxin A and local hyperthermia therapy. However, the effects were temporary and little, until after a hyperpyrexia the PSS was obviously relieved. Based on professional, Botulinum toxin A producing a functional response that generally lasts from 3 to 6 months (Dolly, 2003), and the PSS obviously relieved

over 7 months after injection. Indometacin were used to relieve hyperpyrexia only in one time, as we all know, the action time of the drug was no more than 1 day, so the possibility of relieving due to indometacin in the patient could be excluded. But there's another possible explanation for the effect, relieving of PSS was the natural course of stroke, as we outlined above, a month later, the PSS reappeared, that we can eliminate this possibility.

Clinically, thermotherapy (wax therapy for example) is considered appropriate option for post-stroke patient with spasticity, however, it often provide limited effects for short durations (several hours). As in this case, hyperthermia relieved PSS obviously and lasted for longer (about 1 month). Inspired by this, whole-body hyperthermia may be work better than local thermotherapy, and with different functional mechanisms. Shuji Matsumoto et al. determined the efficacy of thermotherapy for spasticity and found all F-wave factors of tibial nerve were lower post-treatment (Matsumoto et al., 2006) as we all know, F-wave amplitude is an indicator of alpha-neuron-excitability, which was actually altered by increased gamma-motor neuron activity. Hyperthermia play an important role in the decrease of spasticity might through to lower the activity of gamma afferent fibers through a nervous system response, and the relaxing of muscular and soft tissues. To our knowledge, investigation in to the anti-spastic effects of thermotherapy in post-stroke patients was rare. The mechanisms by which hyperthermia alter PSS are likely to be complex. The next step, further study will be necessary to evaluate the feasibility of clinical application of hyperthermia, as an economical and safe anti-spastic therapy for PSS. On the other hand, hyperthermia (natural or artificial) may cause excessive sweaty, long-term hyperthermia may present electrolyte disturbance and circulatory shock. We have to take precautions against negatively influences of hyperthermia.

In conclusion, we report an interesting case of hyperpyrexia caused by upper respiratory tract infection obviously relieved post-stroke spasticity, this interesting phenomenon was meaningful and thought-provoking and the underlying mechanisms should be further studied.

Conflicting of Interests

All authors declare that there is no conflict of interest.

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