Case Report: Effects of Systolic Extinction Therapy in a Patient with Seropositive Rheumatoid Arthritis

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TREATMENT DESIGN. In conjunction with pharmacotherapy (MTX, Sulfasalazine and cortisone), the patient received behavior therapy (OBT), which combines elements of cognitive behavioral therapy (OBT), systemic extinction training (SET), which combines elements of classical conditioning and extinction training, behavioral therapy (OBT) and systemic extinction training (SET), which combines elements of classical conditioning and extinction training, and pain [5], observed as enhanced pain behavior and subjective pain severity in response to solicitous spouse behaviors. The treatment goal is reduction of pain behaviors, lower subjective pain ratings, and increased frequency of healthy adaptive behaviors, such as goal-oriented adaptive physical activities, increased assertiveness in social relationships, and reduced catastrophizing.

Baroreceptor training delivers pain-free and painful stimuli in scheduled sessions, as well as a diminished baroreflex sensitivity (BRS). Patients with a hypertensive reactive to prolonged stress may develop permanently increased blood pressure with reduced variability, which diminishes activity of arterial baroreceptors. Diminished BRS is associated with increased pain in patients with chronic pain. Cardiac-gated baroreceptor training increases blood pressure variability with resultant activation of the BRS and inhibition of sympathetic cardiac and vasomotor neurons by reactivation of dmNTS reflex arcs responsible for the regulation of blood pressure, pain and anxiety.

CLINICAL OUTCOME. After 5 weeks of SET in combination with MTX, Sulfasalazine and cortisone, the patient’s clinical pain was reduced from 70 to 0 (VAS=0-100) and she became pain free for 27 weeks. At 12-month follow-up, she reported a 0-100 pain intensity of 55 for RA and 0 for FM & OA, at the 27-month follow-up 10 for RA and OA.) For FM whereby the cortisone was reduced from 20mg to 5 mg maintained for the following 27 months (Figure 1). The patient has 2 refreshment sessions during the follow-up. Blood pressure became normotensive (92/53 mm/Hg at rest and 112/63 mm/Hg during stress) 12 months after therapy and could be maintained for further 24 months (116/67).

Sleep rhythm normalized, with 32 arousals and 2 awake phases in the 2nd half of the night. BRS increased to 69.1% after SET combined with MTX, Sulfasalazine and cortisone, to 70.34% 12 months and 71.67% 27 months after therapy. Disease Activity Score (DAS28) fell from 5.28 to 2.88 after treatment and remained low (3.14 ± 3.2) at the 27 month-follow-up.

Tender points on examination were no longer detectable. During 27 months, the patient experienced 1 episode of inflammation of the right knee. No further inpatient treatment was necessary.

IMMUNOLOGICAL CHANGES. CRP reduced to 8.7 mg/l after therapy and remained low (14.7 mg/l) 27 months after therapy. IgM and IgG rheumatoid factors were normal at 12 and 27 months. The anti-CCP-antibody reduced to 94.65% and was 81.67% 27 months after SET in combination with MTX, Sulfasalazine and cortisone (Table 1). The pharmacotherapy (MTX and cortisone) decreased the anti CCP-antibodies to 73.89% within the first 3 years before SET was added.

DISCUSSION. It is generally accepted that stimulation of carotid sinus and cardiopulmonary baroreceptor afferents, which are activated by dynamic changes of blood pressure and respiratory rate, reduce pain. Baroreceptors provide signals to the brainstem and the dorsal medial nucleus tractus solitarius (dmNTS). The dmNTS is responsible for inhibition of both pain, anxiety, sleep quality, and blood pressure. In healthy individuals an elevation in resting arterial blood pressure leads to a reduction in acute pain sensitivity. Studies show that in persistent pain conditions the interaction between blood pressure and pain sensitivity is impaired. Diminished baroreceptor sensitivity, which is influenced by classical and operant conditioning, may provoke pain chronification. The persistent stress-related increase of blood pressure and elevated dmNTS activity, as an effect of dynamic changes of arterial baroreceptors, may elicit the carotid sinus. In effect, the carotid baroreceptors “learn” not to react anymore. The NTS no longer receives inhibitory signals and, therefore does not inhibit pain and anxiety.

Diminished NTS activation leads to the development of pain chronicity in the subgroup of patients who develop hypertension in prolonged stress situations. In addition, diminished BRS and NTS activation effects the immune system. Nonpeptidic (NE), through stimulation of the β1-adrenergic receptor-cAMP-protein Kinase A pathway, inhibits the production of type 1 proinflammatory cytokines, such as interleukin (IL-12), tumor necrosis factor-α, and interferon-γ by antigen presenting cells and Th1- and Th17-cells. Through the endogenous catecholamines may cause a selective suppression of Th1 responses.

CONCLUSION. This single case suggests that SET, by restoring BRS and NTS activity, enhances the sympathetic neural system in reducing inflammation and/or pain in RA, OA, and fibromyalgia.

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