Die Hyperbare Sauerstofftherapie (HBO) im

Therapiekonzept

Complex regional Pain Syndrome (CRPS)
Morbus Sudeck
Sympathische Reflexdystrophie

in den Druckkammerzentren

des VDD e.V.

Verband Deutscher
Druckkammerzentren

Zusammenstellung von Informationen

für Ärzte

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Zusammenfassende Beurteilung

Für die Anwendung der hyperbaren Sauerstofftherapie (HBO) beim Morbus Sudeck liegen zahlreiche Fallberichte über die erfolgreiche Anwendung vor. Inzwischen gibt es auch eine kontrollierte Studie, die nach der Evidenz basierten Medizin die Kriterien der höchsten Kategorie (Klasse 1b) für Einzelveröffentlichungen erfüllt.

In Fällen, die mit den üblichen Behandlungsmethoden im multimodalen Anasatz keine ausreichende Verbesserung erfahren ist deshalb die Anwendung der HBO sinnvoll und anzuraten. Die Basisbehandlung muss dabei weitergeführt werden.

Literaturübersicht

Kiralp,-M-Z; Yildiz,-S; Vural,-D; Keskin,-I; Ay,-H; Dursun,-H: Effectiveness of hyperbaric oxygen therapy in the treatment of complex regional pain syndrome. J-Int-Med-Res. 2004 May-Jun; 32(3): 258-62

In this **double-blind, randomized, placebo-controlled** study we aimed to assess the effectiveness of hyperbaric oxygen (HBO) therapy for treating patients with complex regional pain syndrome (CRPS).

Of the 71 patients, 37 were allocated to the HBO group and 34 to the control (normal air) group. Both groups received 15 therapy sessions in a hyperbaric chamber. Pain, oedema and range of motion (ROM) of the wrist were evaluated before treatment, after the 15th treatment session and on day 45.

In the HBO group there was a significant decrease in pain and oedema and a significant increase in the ROM of the wrist. When we compared the two groups, the HBO group had significantly better results with the exception of wrist extension.

In conclusion, HBO is an effective and well-tolerated method for decreasing pain and oedema and increasing the ROM in patients with CRPS.

Peach G.: Hyperbaric oxygen and reflex sympathetic dystrophy: a case report",. Undersea Hyperbaric Medicine 1995; 22(4):407-408.

A patient suffering from acute smoke inhalation also had a long medical history that included reflex sympathetic dystrophy syndrome of the left foot and ankle. The entire foot and ankle were tender and cool to palpation; range of motion was severely reduced. Shewas referred for hyperbaric oxygen therapy , and 15 minutes into the firsttreatment (46 min at 60 swf) she reported a lessening of the pain in her foot; moreover, the foot was less cyanotic and warmer to the touch. Subsequent treatments continued to improve her conditions and for longer periods of time.

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SUMMARY: The decrease in tissue hypoxia obtained with Hyperbaric Oxygenation (HBO2) counteracts the effects of reflex vasomotor disturbances caused by an injury in post-traumatic Sudeck's syndrome. In reflex sympathetic dystrophy, after an initial vasospasm, a loss of vascular tone with persistent vasodilatation causes increased osseous vascularity and rapid bone resorption. Chronic edema results from venous overload and passive capillary repletion. Local lack of oxygen and acidosis cause demineralization and bone protein, atabolism. The hypoxic static induces undifferentiated mesenchymal cells and younger fibroblast to a rapid maturation, with abnormal production of fibrous tissue, retraction, and adhesions and joint stiffness.

In our experience HBO2 proved to be very effective even after a few treatments resolve local swelling and to relieve pain in 13 of 15 patients affected by Sudeck's Syndrome who had not positively reacted to other therapies. In 14 patients the sympathetic dystrophy affected the lower limb. Strict diagnostics criteria based on history, physical examination and radiological pictures have been respected. Technetium scintigraphy was performed and confirmed diagnosis in 7 cases. A second Te scintigraphy carried out after 20 sessions of HBO2 2.5ATA was available in 5 patients and demonstrated normalization of the vascular phase in 4 patients, and amelioration of the late (bone) phase in 3.

Post-traumatic Sudeck's Syndrome is a reflex sympathetic dystrophy which consists of pain and tenderness, usually in a distal extremity, associated with vasomotor instability. swelling and trophic skin changes arising after trauma. The severity of the syndrome is frequently unrelated to the severity of the injury and the dystrophy of often appears after minor trauma. The classic radiographic picture shows acute, patchy bone demineralization. Technetium scintigraphy displays augmented periarticular radionuclide activity. In its early manifestation as Sudeck's Syndrome is unrecognized or misdiagnosed and mistreated in many cases so the patient may have a prolonged and severe disability.

No treatment, hitherto has proved to be very successful, once the disease has become established: various forms of physiotherapy, systemic administration of drugs (anti-inflammatory agents, vasodilators, steroids, calcitonin), peripheral chemical sympathectomy, infiltration of painful areas with local anesthetics, sympathectomy and sympathetic blocks, section of the sensory nerves or of the dorsal roots of the spinothalamic tract (in intractable cases) have been reported in the literature.

Despite any or all of these measures, many patients improve little or not at all, so that their symptoms persist for months or years. Some patients have attempted suicide because of all the psychological and economical problems related to the disease. The etiopathology of the condition is uncertain. The present pathogenic hypothesis is that after an injury to the limb there is an initial vasomotor reflex spasm and, in a second phase, a loss of vascular tone with persistent vasodilatation and rapid bone resorption.

The increased osseous vascularity appears on the radiogram as a mottled rarefaction caused by increased porosity and decrease in size, thickness and number of trabeculae. Chronic irritation of peripheral sensory nerve secondary to trauma and soft tissue damage determines increased afferent input, abnormal activity of internucial neuronal pool and continuous stimulation of sympathetic motor efferent fibers.

Accordingly to the "gate control theory", predominant small fibers input could result in the unchecked transmission of pain through an "open gate" and create the potential for summation, suppressing the influence of the substantia gelatinosa. Capillary bed repletion, venous overload, opening of the arterovenous shunts provoke tissue hypoxia, catabolite formation, chronic edema and acidosis. Acidosis, inactivity and vascular stasis determine bone resorption of the cortical haverslan system. Hypoxia and acidosis lead undifferentiated mesenchymal cells and younger fibroblast to proliferation and quicker maturation (a state which requires lower oxygen consumption) with abnormal fibrous tissue production, edema organization and joint stiffness. Reflex vasomotor disturbances, resulting in hypoxia, catabolite production and acidosis stimulate sensory nerve termination and close a vicious self sustaining cycle.

The use of HBO2 in the treatment of post-traumatic Sudeck's Syndrome is rational. In fact hyperbaric oxygenation induces vasoconstriction and reduce edema: this counteracts vascular stasis and venous repletion, increases depresses osteoblast activity and mineralization, reduces fibrous tissue formation. HBO2 therapy seems to break the vicious self sustaining cycle of reflex sympathetic dystrophy, because normalization of local tissue oxygen tension, pH and water interstitial content stops abnormal sensory nerve stimulation and efferent vasomotor phenomenon's.

MATERIAL AND METHOD: Fifteen patients, (11 men and 4 women) suffering for reflex post - traumatic dystrophy have been treated with HBO2 therapy. In 14 of the 15 cases the trauma affected the lower Limbs. The average age was 44.4 years. Initial injury was in 4 cases a calcaneus fracture In 3 cases a malleolus fracture; in the remaining patients Sudeck's Syndrome followed tibial shaft fracture (2 cases), supracondylar femur fracture, multiple metatarsal bone fractures, multiple metacarpal bone fractures and in 3 cases only an history of minor trauma was collected. The disease involved foot | and ankle in 13 cases, the knee in one case and the- hand and the wrist in no case. 10 patients had immobilization ion in cast as the treatment of choice in 3 cases (supracondylar femur fracture, multiple metacarpal bone fractures, malleolus fracture) the patient underwent surgical treatment. Time elapsed between trauma and diagnosis was 2- 8 months.

Strict diagnostic criteria for inclusion in the study hen been based on history of injury to an extremity, basic examination and radiological picture. Technetium scintigraphy was performed in 7 cases to confirm diagnosis and in 6 cases assessed the evolution of the disease. Clinical diagnosis was based on the presence of pain, tenderness, swelling, vasomotor instability and joint stiffness long lasting after a trauma. Radiographic criteria included patchy. bone demineralization, osteoporosis and cortical cavitation. All the patients were in the acute phase of the syndrome. No case of treatment of the initial or of the atrophic stage has been included in the present study. HBO2 protocol consisted in 20 sessions at 2.5 ATA ((5 sessions A week). A further series of 10 sessions was performed in patients (3 cases) present partial clinical recurrence during the week ensuing the termination of the 20 session protocol. A previous calcitonin regimen, although of very limited efficacy, was maintained during HBO2 therapy in 5 subjects. No patient used analgesic drugs during HBO2 treatment. Avoidance from weight bearing, functional limb rest and use of an elastic stocking were strongly counseled in patients with lower limb involvement. Te scintigraphy was performed at the end of the 20 HBO2 sessions in 6 cases. Radiographic controls were scheduled at 2 and 4 months.

RESULTS: After the first week of HBO2 a marked reduction of pain and tenderness in the extremity was observed in 9 patients: discrete clinical improvement has been recorded in 3 cases. Reduction of swelling and restoring of movements in the affected extremity has been progressive during the course of HBO2 therapy. At the completion of the first HBO2 cycle complete recovery (no pain complete restoration of movements in the affected joints, no swelling) has been observed in 4 cases. Marked clinical improvement (occasional light pain minimal swelling at the evening, almost normal movements in the affected joints) was present in 5 cases. Moderate clinical improvement (reduction of pain and swelling partial restoration of movements) has been present in 4 cases. In 2 patients despite some reduction of swelling significant pain persisted, in one of these patients, however, pain was present only during weight bearing on the affected extremity and in part could be referred to progressive subtalar degenerative changes after a calcaneus fracture. In 4 cases partial relapse of the symptoms in the weeks ensuing the completion of the first 20 HBO2 sessions lead to a second 10 session HBO2 cycle with complete recovery. In the 6 cases controlled at the Te scintigraphy after the 20 HBO2 sessions normalization of the vascular phase was observed in 4 patients, and reduction in the hypercaptation in the late (bony) scintigram was present in 3 cases. No case of worsening of the scintigraphic picture has been recorded. Resolution of the classic radiologic pattern has been generally slow: In a few patients significant improvement at the 2 month control has been observed.

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