Hyperbaric Oxygen in the Treatment of Sudeck's Syndrome

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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 ln 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.

ILLUSTRATIVE CASE REPORTS
1) A 50 year old bricklayer sustained a sprain to his left ankle which remained untreated. After two months ankle pain. quite slight at the beginning, get increasing with paroxysmal exacerbations ,extending to the forefoot and forcing the patient to suspend his work. The radiogram showed the classical picture of reflex sympathetic dystrophy. Pharmacological agents and physiotherapy remained for months ineffective. Presenting to our observation,6 months after the injury, the patient was unable to walk without crutches, suffered of intense and unduly pain and was severely depressed, lacking of confidence in any form of treatment. Clinical examination revealed minimal swelling of the ankle, cutaneous hypersensitvity and a 50% decrease in movement of the subtalar and tibiotalar. After the first week of HBO2 therapy the patient referred significant decrease in pain which after the second week almost disappeared. A progressive and

complete recovery of the movements of the joints involved was recorded. After 20 sessions of HBO2 patient was free of any symptom and walked normally. Te scintigraphy demonstrate normalization of the vascular phase and clear reduction of hypercapration in the late phase. Resolution of radiographic picture was slow.

2) 58 year old man. pensioner after an untreated left fore foot distortion the patient complained persistent refractory pain swelling, limitation of motion in the extremity and marked disability to walk. On the basis of clinical radiologic and To scintigraphic findings diagnosis of reflex algodystrophy was formulated 5 months after trauma. After only four HBO2 treatments pain and swelling disappeared at the completion of the schedule the patient walked correctly without crutches and was very satisfied. Te scintigraphy at the end of the therapy demonstrated significant reduction in the hypercaptation of the forefoot. At the 2 month control discrete amelioration in the radiologic pattern was observed.

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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