SÜDECK’S POST-TRAUMATIC OSTEODYSTROPHY

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SÜDECK’S POST-TRAUMATIC OSTEODYSTROPHY (Abstract): The diversity that exists in the types of trauma, in the investigated anatomical structures, in the sites of trauma, in the outcomes of traumatic injuries, and in the general reaction of the body lead to a symptomatical polymorphism of post-traumatic sequelae. Therefore it is impossible to establish a well-defined nosological entity of these sequelae. The damaged tissues react under a very similar scheme, irrespective of the type of tissue, trauma or area, namely, through an inflammatory process, that causes various sequelae depending on certain parameters. Südeck post-traumatic osteodystrophy may be defined as a pathological entity based on some well-defined clinical features, on the development of its own therapy and on its more or less ubiquitous character. Südeck’s post-traumatic osteodystrophy of the hand is rarely found isolated. This "acute bone atrophy" is usually included in the "pathological syndrome of the hand" and is caused by circulatory disorders that occur on a traumatized hand or on a hand with injury "at a distance", especially when nerve damage is involved. Keywords: ALGONEURODYSTROPHY, POST-TRAUMATIC SEQUELAE, REFLEX SYMPATHETIC DYSTROPHY.

Vandenabelle (1) gave in 1976 an historical overview of algoneurodystrophy (AND) including description, outcomes and the names under which this disease was known: post-traumatic osteoarthropathy, acute bone atrophy, post-traumatic painful osteoporosis, reflex sympathetic dystrophy, neurotrophic rheumatism, shoulder-hand syndrome, Südeck-Leriche syndrome, neurotrophic syndrome, sympathetic algodystrophy of the limbs). Etiopathogenic and pathophysiological mechanisms were also chronologically reviewed.

Algoneurodystrophy is a form of disease that has a great variety of clinical aspects (2). That is why there are many controversies surrounding the different issues that define its nosological relations. One of them is “the acute inflammatory bone atrophy” (1900, Südeck) (3), which was seen as a clinically well-defined post-traumatic sequel. The anatomical substrate is the osteoporosis, that was called "acute inflammatory bone atrophy" by Südeck and was seen as a clinically well-defined post-traumatic sequela, but Südeck considered actually the inflammatory process as dominant.

The first relatively complete anatomoclinical description of algoneurodystrophy is attributed to Südeck (1900). The term “anatomical substrate” was used
for the first time by Südeck and this was the premise for describing the radiological changes. The anatomical substrate is the osteoporosis that was called "acute inflammatory bone atrophy" by Südeck and was seen as a clinically well-defined post-traumatic sequela.

This disease was later called "post-traumatic painful osteoporosis" by Leriche (3). He considered it as a local disturbance of the neurovegetative system and, as the sympathetic nervous system plays a main role, it was also called "reflex sympathetic dystrophy" in the Anglo-Saxon speaking world. The disease was later known as Südeck-Leriche syndrome (3).

Some neurotrophic disturbances may occur at the level of the hand after the injuries of this segment, as well as after the injuries "at a distance" of the upper limb (4).

These disturbances are not consistent with the severity of the primary traumatic lesions, but rather with the subsequent complaints experienced by the injured person, with the excitability of his nervous system, with the administered treatment and with the poor outcome of treatment of lesions.

Definition of Südeck’s post-traumatic osteodystrophy. Osteodystrophy is a generalized disease of the skeleton, characterized by decreased bone mass and deteriorated micro-architecture of the bone tissue. Osteoporosis leads to increased bone fragility and risk of fractures that may occur in minor trauma or even spontaneously. Osteoporosis is the most frequent metabolic bone disease and affects women more than men. It decreases the bone strength, which brings a greater risk of fractures. The major issue in the management of osteoporosis lies in the prophylaxis of this disorder, and the most economically advantageous and the easiest to apply method in the patient self-care is the kinetotherapy prophylaxis.

Pathophysiological syndrome of the hand. The disorders of this syndrome may be designated as "syndrome of post-traumatic nervous irritation" or as "post-traumatic reactive syndrome" (5).

These post-traumatic trophoneurotic disturbances of the upper limb and of the hand in particular represent a disputed issue, both in terms of etiopathogenesis and treatment. The trophic disturbances have a progressive onset, from fingers to shoulder, when the lesion is distal, and from top to bottom, when the lesion is situated at distance from hand. No tissue is spared and there are to be seen alterations of the structure of skin, hair, nails, subcutaneous cellular tissue, aponeuroses, muscles and bones. The affected hand does not look like a normal hand anymore (6).

In milder cases, the tissue alterations suggest a simple atrophy caused by inactivity. In other cases, the extent of tissue alterations, the rapidity of their development, the associated vasomotor and motor disturbances, as well as the unusually high degree of function loss are due to the dynamic processes.

The pathophysiological syndrome of the hand may take various forms, depending on its intensity: from the simple atrophy caused by inactivity to the complex forms, associated with acute pain. The nervous mechanism that acts on the veins, capillaries, arteries and arterioles, causing nutritional disturbances in all tissues of distal segment of the upper limb, triggers also most of the alterations. Once this mechanism has been activated, it usually maintains a hyperemia with venous stasis, that
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depthly disturbs the trophicity of the tissues as it becomes chronic.

The physiopathic syndrome of the hand is always associated with severe forms of Südeck’s osteoporosis. Most often the symptoms of these syndromes overlap and may be confounded: persistent pain that is exaggerated as compared to intensity of trauma, loss of hand function, edema and atrophy of subcutaneous cellular tissue, trophic disturbances of the skin, severe bone demineralization.

The topography of pain and of hypersesthesia does not correspond to the territory of any somatic nerve, as it is less localized and it is unbearable "as a visceral pain". The lesions of arteries or the injuries of the median nerve predispose frequently to nerve irritation syndrome; on the other hand, the median nerve has a much larger number of fibers that travel to the blood vessels as compared to the radial or the cubital nerve.

The centrifugal impulses that origin in the central nervous system and travel to the muscles, sudoriferous glands and blood vessels, as well as those that control vital activities of tissue cells are continuously dependent on the centripetal impulses coming from periphery. If these afferent messages are normal, then the efferent impulses are also normal. But if the afferent messages develop a harmful intensity and cause a permanent irritation, they disrupt the harmony of this automatic regulation, and the efferent impulses become also pathological and generate various disturbances (motor and vasomotor disturbances).

The cortex plays an important role in all these neurovascular disorders. In persons with labile nervous system, the nociceptive stimuli that come from the site of trauma through exteroceptors and proprioceptors act employing the cortex on the neurovascular system of the upper limb and of the hand in particular. This system is disturbed for a more or less long period of time, but sometimes it is chronically disturbed.

Any trauma, no matter how mild it may be, generates an irritative focus, that acts on the surrounding proprioceptors, that are very well developed at the level of fingers and in hand joints. The impulses that come from these areas and travel to the cerebral cortex generate changes in brain dynamics. If the signals that originate in the proprioceptors coincide in time with the excitations of the exteroceptors, conditioned reflexes can be produced. These conditioned reflexes may be sometimes fixed and lasting and may exert multiple effects on the function and trophicity of the tissues.

Clinical aspects. The symptomatology shows three clinical stages of evolution. Stage 1 emerges immediately or sometimes within a few weeks after the injury. It is characterized by acute pain, that increases with movement, skin hyperemia (the skin is warmer and more moist than normal), muscular hypotonia, edema of skin, of connective tissue and of muscle, early osteoporosis (7). Stage 2 develops in the next three months. The pain persists, the skin is cyanotic, cold, moist, and the hair in the area breaks off or gets lost. The edemas persist, joint stiffness occurs and the radiographic diagnosis is represented by the characteristic spotted osteoporosis. Stage 3 is generally regarded as irreversible and it is characterized by atrophic processes of skin, muscles, by having aponeurotic retraction and tendon retraction, severe osteoporosis (in some cases marked bone resorption), functional impotence of joints. At this stage the pain is
decreased, but it disseminates to the limb root. The skin becomes dry and cold.

Hyperemia may explain the osteoporosis, the edema of soft tissues, but it cannot fully motivate the pain, the atrophy and the sclerosis.

Thus, the syndrome is clinically characterized by hyperemia and cyanosis of the skin, that are usually associated with subcutaneous edema and with impairment of finger movements. The joint stiffness and the muscle atrophy also occur. The persistence of pain, the hard edem, the atrophy of subcutaneous tissue and of muscles, the thickening of the joint capsule, the deformation of the fingers in flexion, the progression of osteoporosis may also indicate this syndrome.

The evolution shows various forms of onset. Acute onset – in full health, weeks and rarely months after a traumatic episode or after an organic disorder, an algoneurodystrophic syndrome and its characteristic symptoms develop in 24 hours or in 30 days at most. In the cases with acute onset, the local symptoms are very intense and the general repercussion on the body is significant. Subacute onset – is the most common form. In 6-7 days the painful and vasomotor syndrome occurs. The local and general symptoms are less obvious than in the acute dramatic form of the acute onset. Chronic onset – is found usually at an older age, in neurological patients and in less reactive persons. In 15 days at most a painful and vasomotor syndrome gradually emerges, but it is frequently attenuated and therefore difficult to diagnose (8).

**Diagnosis.** The inflammation tests are negative and ESR may have values of 30 mm/h. The radiography reveals a local demineralization and shows a spotted osteoporosis of spongiosa. The compact bone of the diaphyses is spared for some time, but then it also become thin. Nevertheless joint space narrowing or cartilage thinning are not found.

The bone scintigraphy shows characteristic uniform uptake. If the traumatic lesions are peripheral (fingers), the atrophy progresses in a centripetal direction. If the lesions are "at distance" from hand (e.g. lesions of the nervous trunks), the bone atrophy progresses in a centrifugal direction, towards the periphery of the upper limb.

Osteoporosis occurs in closed or open mechanical trauma, in burns, frostbites, nerve damage and especially in the latent inflammations that follow these injuries.

The histopathological examination shows a discrete synovial and subsynovial edema. There are also perivascular cellular infiltrations with lymphocyte predominance.

**The treatment** is developed with regard to the triggering event, to the severity of trauma, to the stage of disease and to the prophylaxis.

If the syndrome begins to develop, the treatment involves the suppressing of all irritations that originate in the traumatic focus, balancing the nervous system, recovering the normal activity of the hand (active kinetotherapy).

The suppression of irritations is made by complementing the surgery of traumatic focuses and of their complications, namely, unreduced fractures, non-eliminated sequestra, irritating foreign bodies, painful nerve injuries, subacute infections, etc.

One of the best means of suppressing irritations of the traumatic focuses is the proper immobilization of the injured segment (immobilization depending on posi-
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tion The balancing of the nervous system is also important and it is achieved by gaining patient’s confidence and by obtaining the patient’s full compliance to treatment, as well as through novocaine infiltrations at various levels, especially on the upper thoracic ganglia.

The somatic nerve infiltrations have a good outcome. They may be performed at distance from hand, on the nervous trunks or even on the brachial plexus or they may be locally applied, around the lesion, in the area of hyperesthesia. Thus, they act for the suppression of the nociceptive irritations. Local infiltrations with novocaine have a better outcome when the point from where the afferent nociceptive stimuli originate is precisely identified and injected. The infiltrations on the regional sympathetic nervous system act on the afferent pathways.

The recovery of the normal activity of hand (when possible) leads to healing in the shortest possible time. All procedures should be always performed carefully, skillfully and with full compliance of the patient, in order to achieve simultaneously the immobilization needed for the suppression of the irritations of the traumatic focus and the active mobilization of the uninjured segments. This mobilization should be performed carefully and persistently, progressively, as the movements must always be made below the threshold of pain.

The concomitant upper extremity suspension that reduces the venous stasis and edema, contributes to the implementation of the active kinetotherapy.

The edema of the back of the hand may represent one of the components of the physiopathic syndrome, especially when this edema becomes chronic as in hard edemas. However, the chronic edema of the back of the hand may also occur isolated, without the other components of the syndrome or without having components so marked as the edema. The edema requires surgery when it becomes chronic and hard.

Curative treatment – has the following aims: to eliminate pain, to intercept the pathogenic line, to recover the function of the affected segment. Combining drug therapy with kinetotherapy and with orthopedic surgery may result in cutting off the reflex circle, the restoring of a normal active vascularization and not least the pain relief.

The therapeutic means that pursue the specific blockade of the sympathetic nervous system represent the most reasonable strategy. This blockade alleviates pain, because the pain impulses that are produced as a reflex in the somatic C-fibers by the excitation of the adjacent sympathetic postganglionic fibers disappear. Thus, the pathological afferentation towards the intercalary neurons is interrupted (or reduced) and results in the reduced activity of these neurons. But the blockade does not completely eliminate pain, because the pain impulses continue to be triggered from the hyperemia area of the affected joints and they travel through the common thick somatic fibers towards the spinothalamic pathway.

Vasodilators are frequently used. Calcitonin reduces bone resorption, decreases the activity and the number of osteoclasts, but they do not repair what has been lost. It also decreases rapidly the pseudo-inflammatory phenomena and it has an analgesic effect through a double mechanism (peripheral and central). Calcium preparations, synthetic anabolic substances and psychotropic drugs may also be used.

The kinetotherapy treatment aims to
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restore joint mobility and the muscle tone and represents the mainstay of recovery, but when associated with physiotherapy can give in some cases even better results (1, 10). Through the used methods and procedures, kinetotherapy plays a very important role both in primary and in secondary prevention, as the use of a specially structured and assembled physical exercise has a beneficial effect on disease prevention, as well as on prevention of deficiencies and dysmorphisms caused by sequelae of medical conditions or injuries (9).

Südeck and Böhler (cit. 10) suggested that this syndrome may be prevented if the nociceptive stimuli are suppressed. Although bone atrophy is thought to be "inevitable" in risk populations, the complete syndrome develops only if the rules of proper immobilization of the traumatic focus are not followed. This immobilization should be associated with the active mobilization of all uninjured segments. "No mobilization exercise should cause pain", because the pain is the most important nociceptive stimulus in such cases. This stimulus leads to neurovascular disturbances that maintain or aggravate Südeck's syndrome. If the syndrome is installed (at the stage of spotted atrophy of spongiosa), the safest means of stopping its development is the proper immobilization itself, namely, uninterrupted, in the passive posture of the affected segment, with the active, careful mobilization of the mobilized segments, as long as the pain does not arise (11).

In order to combat the venous stasis and the edema, the suspension of the upper limb or at least of the hand can be simultaneously used. The following physiotherapeutic measures may also be recommended for accelerating the healing: short warm baths (for 10 minutes) followed by the suspension of the upper limb, circulatory gymnastics through warm baths alternating with cold baths, cold air baths (for 15-20 minutes), and diathermy may also have beneficial effects (12).

In bone atrophies with finger stiffness, the perseverance of kinetotherapists and the full and conscious compliance of the patient may result in the partial recovery of finger motility (13).

**Prognosis of Südeck's syndrome.** The post-traumatic Südeck's syndrome has usually a favorable prognosis, as the acute bone atrophy disappears progressively after the suppression of irritative noxae.

If the bone atrophy persists as a result of maintain the continuous irritations, it may become chronic and a marked osteoporosis of the whole upper limb develops. These bones have a "glassy" appearance.

The acute "spotted" atrophy may develop into chronic bone atrophy if the primary lesion and the irritations that arise from it are not managed in time.

The evolution of the disease is unpredictable, and remains unpredictable even when a complex pathogenic treatment is administered. Many forms that were treated with complex therapies evolve arbitrarily, and the vasomotor and painful symptoms may return after they have regressed as a result of the treatment (14, 15, 16).

**CONCLUSIONS**

The best management of Südeck's disease is represented by the consideration for improving of the kinetotherapeutic strategies, for pain relief, for reducing vascular stasis, for avoiding contractures and capsular retractions, as well as for combating anxiety and depressive moods in patients.
By means of an educational therapeutic rehabilitating process, kinetotherapy aims to improve the physical and mental health of persons with special health needs, in order to facilitate them the social and occupational integration or reintegration.

REFERENCES