Post-traumatic Sympathetic Dystrophy

—A Case Report—

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A case of post-traumatic sympathetic dystrophy following crushing injury to the left fingers is described. A brachial angiogram showed obliteration of the radial artery at the level of the wrist joint. Following dorsal sympathectomy, the persistent pain was relieved and trophic changes disappeared.

Pain in an extremity that develops following trauma, infection, thrombophlebitis and many other lesions has been recognized for many years and designated by a variety of names. Mitchel (1959) gave a classical description of a syndrome concerning pain produced by nerve wounds. In Sudeck (1959)'s original paper many descriptions of post-traumatic painful extremities have appeared. Such terms as Sudeck's atrophy, Sudeck's syndrome, minor causalgia, causalgia-like state, post-traumatic painful osteoporosis, reflex dystrophy of the extremities, post-traumatic dystrophy and sympathetic dystrophy have been used to designate this syndrome. Sympathetic dystrophy is considered to be a causalgic state of an extremity relieved by sympathetic interruption. It differs from true causalgia in that no demonstrable peripheral nerve injury has occurred (Bergan and Conn, 1968). Thus the post-traumatic dystrophies include all of the conditions termed by Homans (1940) as minor causalgias. Sudeck's atrophy refers to the characteristic osteoporosis that frequently occurs in causalgic states.

We report one case of post-traumatic sympathetic dystrophy and review literatures.

CASE SUMMARY

This 42 year old man was admitted on Oct. 13th, 1976, due to severe pain in his left hand following a crushing injury at work to the left middle finger by a metallic hammer.

He suffered the above injury on Apr. 1, 1976. After that he was admitted to a private hospital. At that time his left middle finger tip was found crushed, so debridment was performed.

In spite of this, the wound became infected and pus had to be drained out. He was transferred to the Orthopedic Department of Severance Hospital where the wound in his left middle finger healed, but from September, 1976 he suffered from severe episodes of burning pain with numbness in his left middle, 4th and little fingers, especially when these fingers were exposed to cold. Because of the intractable pain, nerve blocks were tried, but
without any improvement, so he was transferred to the Neurosurgical Department.

On physical examination there was bluish discolorization of the middle, ring and little fingers of the left hand, but no palpable radial pulsation (Fig. 1). X-ray showed no abnormal-

![Fig. 1. Preoperative photograph of patient's hands. Bluish discolorization of the left hand compared with the right hand.](image)

![Fig. 2. Postoperative photograph of patient's hands. The color became same.](image)

ities. Left brachial arteriogram showed obliteration of the radial artery at the level of the wrist joint (Fig. 3). Skin temperature of the left fingers was 6°C lower than that of the right fingers.

Left dorsal (T2, 3) sympathectomy was done on Oct. 15, 1976. Postoperatively, skin color became normal and the skin temperature of the left side was the same as on the right

![Fig. 3. Brachial angiogram showing tapering off of the left radial artery.](image)

(Fig. 2). The patient did not complain of any pain.

**DISCUSSION**

More than two centuries ago Pott described the pain which followed partial division of a peripheral nerve. Bell in 1812, Scarpa in 1832 and Hamilton in 1838 reported on patients with painful extremities following partial injury of nerves (Drucker, *et al.*, 1959). In 1864 Mitchell, Morehous and Keen, who were stationed at a military hospital, gave a classic description of a syndrome concerning pain produced by nerve wounds. In 1900 Sudeck described acute atrophy of bone in the extremity which he ascribed to inflammation.

During the fifty-nine years that have elapsed since Sudeck's original paper many descriptions of post-traumatic painful extremities have appeared. In 1940 the post-traumatic dystrophies include all of the conditions termed by Homans as minor causalgias. In 1948 Holden pointed out that post-traumatic sympathetic dystrophy differed anatomically from Mitchell's causalgia only in that there was no demonstrable injury to a major peripheral nerve. Miller and de Takats in 1942 gathered up under the
heading of post-traumatic reflex dystrophy a whole series of states named reflex atrophy, Sudeck's atrophy, post-traumatic osteoporosis, traumatic angiospasm, chronic traumatic edema, peripheral trophiculosis and reflex nervous dystrophy. They also pointed out that rheumatic arthritis with its vascular phenomena, the frozen shoulder after myocardial infarction, and the major causalgias have an identical, three stage course, although the clinical picture admittedly differs in intensity and prognosis (Miller and deTakats, 1942).

In 1969 White and Sweet called such a condition post-traumatic arthritis.

Sympathetic dystrophy has many "causes" or precipitating agents. Reme (1956) states that a recognizable cause always exists, whereas Schröter (1959) reported five cases without antecedent injury in which the only trauma was excessive use of the hand.

Drucker (Drucker, et al., 1959) gathered sixty-one cases and analyzed the types of injury and the frequency of occurrence. Fracture was the most common cause. Sprain, laceration and trauma to soft tissues were the next most common causes. Coney (Coney and Enderoff, 1969) gathered thirty-five cases of post-traumatic vasomotor disturbance and analyzed types of injury: twisting injury, compression-crushing, direct trauma and postoperative state. Our case had no fracture and the major injury was compression-crushing with infection.

Bone change was absent in sympathetic dystrophy, but in Sudeck's atrophy there was osteoporotic bony change, and that was refined by Sudeck (1900). He described three types of post-traumatic atrophy: acute atrophy was considered to be a normal physiological sequence of a severe injury to a limb; chronic atrophy was considered a consequence of delayed healing of the primary injury, particularly fracture; chronic traumatic dystrophy was thought to follow apparently insignificant injuries. However it is unlikely that the osteoporosis is a cause of the syndrome. Whether the abnormalities in bone are characteristic or simply a reflection of disuse resulting from painful extremities remains to be settled (Drucker, et al., 1959).

For many years the theory was advanced that the pain and dystrophic changes were a result of altered peripheral blood flow due to sympathetic activity (Drucker, et al.). Many instances of increased blood flow in the involved limb were recorded. On the other hand, both Holden (1948) and Mayfield (1945) demonstrated that the patients may have increased, normal or decreased blood flow. In our case there was definite narrowing of the distal part of the radial artery. One author reported that in the late stages of the syndrome marked vasospasm is frequently present (Hartly, 1955). A satisfactory correlation remains to be established in cases of sympathetic dystrophy between osteoporosis and decreased blood flow.

The problem that faces one who attempts to explain the pathogenesis of sympathetic dystrophy is how a trivial injury can produce pain which far surpasses that of the initial injury and which persists long after the injury has healed. Livingstone (1943) postulated that three factors are involved in establishing a vicious circle of reflexes. The initial factor is trauma and tissue damage resulting in chronic irritation of a peripheral sensory nerve. The chronic peripheral nerve irritation produces an increased number of afferent impulses to the spinal cord. Livingstone's second factor proposes an abnormal state of activity in the internuncial neuron center produced by the increased afferent impulses. An increased activity in the internuncial pool may lead to Livingstone's third factor,
i.e., a continuous and increased stimulation of efferent motor and sympathetic neurons.

Drucker (1959) described the pathogenesis of post-traumatic sympathetic dystrophy well. He stated that the injury causes an increased number of afferent impulses transmitted by large somatic fibers and small unmyelinated C fibers. It also produces an ephaphe so that efferent vasomotor, pilomotor and sudomotor small unmyelinated C fibers, which are part of the sympathetic nervous system, cause a cross stimulation of adjacent somatic afferent fibers.

His description of the second link in the chain is increased activity in the internuncial neuron center produced by the augmented number of afferent impulses. Cerebral and hypothalamic impulses may either inhibit or stimulate the internuncial pool activity. Noxious visual and auditory stimuli as well as increased emotional tension will accentuate the pain in a causalgic state, whereas sleep, a quiet stable environment and narcosis will ameliorate it.

The third link in the chain is activity of the efferent sympathetic nervous system which may produce a variety of dystrophic disturbances in the periphery as well as discharges across the ephaphe to the afferent sensory system.

The forth link which closes the chain is the ephaphe produced by the initial injury. And he also described that an external environmental factor such as temperature may modify the sympathetic activity.

The symptoms of post-traumatic sympathetic dystrophy are pain, swelling, hyperesthesia and hypersensitivity to cold. Treatment of this disease is thus elimination of the above chains, and some methods of conservative treatment are successful, but if the disease progress, sympathectomy cannot relieve the pain (Atkinson, 1975; Holden, 1948). Therefore deTakats pointed out that early treatment of these syndromes is important (deTakats, 1965).

In our case sympathectomy relieved the pain, but about seven days later the patient complained of numbness in his left hand. However with a placebo injection the above complaints disappeared.

**SUMMARY**

A case of post-traumatic sympathetic dystrophy has been described. This considered to be fundamentally the same entity as causalgia. The only significant difference between them is that causalgia is initiated by an injury to a mixed peripheral nerve whereas in sympathetic dystrophy there may have been no such demonstrable injury. The term, Sudeck's atrophy, should be reserved to describe the osteoporosis occasionally found in causalgic states. The pathogenesis of causalgic states is represented as a vicious circle of reflexes under the modifying influence of hypothalamic and environment factors.

This type of pain which is unresponsive to a short trial of conservative measures should be treated by sympathetic denervation of the involved extremity.

**REFERENCES**


