RSDS
Reflex Sympathetic Dystrophy Syndrome
By: Ellen G. Wattay, Ph.D., PT

Physicians are becoming more familiar with the symptoms of reflex sympathetic dystrophy syndrome (RSDS), but there are still many health care professionals who, when they see no objective signs of tissue injury or believe that a patient has had adequate medical and therapeutic intervention but still complains of pain, tell the patient that nothing further can be done. If a patient reports one or a variety of seemingly bizarre symptoms of swelling, temperature changes in the involved region, burning pain, muscle pain, or spasm limiting motor function, atrophy, and skin changes in the area concerned, he or she too often is dismissed as having a psychological problem. The patient is told to see a psychiatrist or that “you must learn to live with it" but is not told how.

Symptoms

Reflex sympathetic dystrophy syndrome was described in the literature over a century ago by Mitchell, Moorehouse, and Keen but remains poorly understood and frequently unrecognized clinically. According to literature provided by the RSDS Association, RSDS generally involves three stages:

Stage 1

1. Onset of severe, burning pain closely limited to the site of injury.
2. Hyperasthesia.
3. Localized edema.
4. Muscle spasm.
5. Stiffness and limited mobility.
6. Vasospasms. At onset, skin is usually warm, red, and dry and then changes to cyanotic, cold, and sweaty.
7. Hyperhydration
8. Average duration of Stage1 is three months. In mild cases, this stage lasts a few weeks and then subsides spontaneously or responds rapidly to treatment.

Stage 2

1. Pain becomes more severe and diffuse.
2. Edema spreads and changes from a soft to brawny type.
3. Hair becomes scant, and nails become brittle, cracked, and heavily grooved.
4. Spotty osteoporosis occurs early but may become severe and diffuse.
5. Increased thickness of the joint.
6. Muscle wasting.
7. Stage 2 may last from three to six months.

Stage 3

1. Marked trophic changes eventually become irreversible.
2. Pain may become intractable and involve the entire limb.

3. Atrophy of the muscles.
4. Interphalangeal and other joints of the foot or hand become extremely weak, have limited motion, and may finally become ankylosed.
5. Contraction of the flexor tendons occurs, and occasionally subluxations are produced.
6. Bone deossification is marked and diffuse.

Etiology
The etiology of RSDS is not yet, well understood. Surgery, trauma, infections, cerebral lesions, spinal and spinal cord dysfunction, and ischemic heart disease are frequently defined as the primary causes, but one third or more of patients with RSDS have no definitive precipitating factors.

Several current theories of the pathogenesis of RSDS exist. One theory is that initial trauma incurs tissue damage, resulting in chronic irritation of a peripheral sensory nerve. This produces an increased number of afferent pulses to the spinal cord and sets up a normal sympathetic reflex arc to any painful stimuli. No temporary vasoconstriction of small vessels occurs. If the sympathetic arc does not shut down as it usually does, an abnormal sympathetic reflex may result. The most widely accepted neurological explanation of RSDS is that a painful stimulus enters the spinal cord via the afferent nerve fibers and stimulates the internuncial pool. Interconnection neurons spread the stimulus upward, downward, and across the spinal cord in short and long circuits, stimulating the lateral and anterior tracts. Efferent autonomic stimulation then reaches the peripheral tissues, producing local circulatory disturbances and muscle spasms that add to the already-noxious stimuli. This produces the so-called "vicious cycle." The increase in activity produces a continuous and increased stimulation of afferent motor and sympathetic neurons, resulting in various responses in the periphery.

J.L. Pool suggested that intensive sympathetic nerve-fiber stimulation might produce a pain-evoking substance. Chapman produced such a substance, which he named neurokinin, by electrically stimulating the distal end of a divided nerve. Neurokinin is a mediator of neurogenic vasodilation of the skin, and, when injected subcutaneously, it produces a flare reaction that lowers the pain threshold in the injected area.

Another proposed factor in RSDS is the concept of artificial synapse. Trauma to a peripheral nerve can cause a "short-circuit" effect. Autonomic impulses from the vasomotor, pilomotor, and sudomotor discharge are augmented by temperature changes and emotions. The peculiar qualities of causalgic pain are ascribed to direct cross-stimulation of sensory fibers by efferent sympathetic impulses at the point of short circuit. This would explain not only the increase in pain that occurs with temperature changes, but also the peculiar exacerbations during sleep, when the tonic hypothalamic discharge is greatly diminished.

Diagnosing RSDS is not as difficult as teaching health care providers to be more aware of and to listen to patients more carefully. Instead of dismissing the patient with RSDS as a "head case," health care providers should look further with thermographic and thermal biofeedback studies, histologic studies of the synovial tissue, and sympathetic block studies. No one determining test or clear-cut classical symptomatology exists.

Treatment

Treatment will vary according to the patient's individual symptoms and history. A team effort of drug therapy, nerve blocks, and physical therapy (including TENS and biofeedback) with close communication among all health care providers is most beneficial. Primary treatment is a nerve block, performed by an anesthesiologist working in conjunction with a primary-care physician and physical therapists. A nerve block is performed by injecting a local anesthetic at the stellate ganglia in the cervical paravertebral area for upper extremity symptoms and in the lumbar paravertebral area for lower extremity symptoms. Physical therapy includes anti-inflammatory or pain-reduction modalities. High-voltage electrical stimulation (probe technique) may be applied to the injected area, along with treatment of associated pain triggers and heat, ice, or ultrasound therapy. Use the patient's response as a guide. Drugs (i.e., anti-inflammatory or pain-killer medications) may be used by the primary-care physician to help relieve symptoms.

If the patient with RSDS is not diagnosed early, a similar team action may be followed up with psychiatric care, antidepressant medications, and techniques to relieve intractable pain (e.g., biofeedback, TENS). The key to major relief is early diagnosis.

Research continues to provide health care professionals with more knowledge of the central nervous system and the total interaction of associated anatomical pathways of nervous system fibers. More neural transmitters and blockers are being discovered, and experiments are resulting in answers to many questions about how we function and respond. In the presentation of RSDS, early diagnosis and therapeutic intervention will give patients the best possible chance of complete recovery or recovery to the point of coping and living a more normal life.

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SUGGESTED READING

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