Reflex Sympathetic Dystrophy: A Case Report and Review

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On hand of a case report, reflex sympathetic dystrophy (RSD) and the related entity of causalgia are extensively discussed in relation to the combat readiness of soldiers.

Reflex sympathetic dystrophy (RSD) is a disease of the sympathetic nervous system which leaves the patient afflicted with a useless limb, intractable pain, and a dysfunctional lifestyle. The pathophysiology of the disease is not well defined. The treatment is centered around resetting the sympathetic nervous system with local anesthetic blockade before the changes become permanent. It is important for the military physician to be familiar with the history and presentation of RSD, in order to make a timely diagnosis and ensure appropriate treatment. This is a disease in which battlefield injuries seem to be a common predisposing factor. If the physician, initially treating the patient, is able to refer him for appropriate therapy, the chances of retaining the soldier on active duty are good.

Case Report

While loading a truck, a 20-year-old soldier, attached to an advanced line unit in Desert Storm, had his foot run over. Initially, there was minimal pain, and radiographic examination did not demonstrate any fractures. The soldier returned to duty. Approximately one week later his foot had swollen and was very sore when he put on his boot. It was felt that he had a severe sprain of the ankle, and he was returned to duty. The swelling continued, and the pain became much worse. The soldier tried to treat the swelling by keeping his boot tightly laced. It became obvious that he was losing function of the extremity. The patient presented to his medical facility and was immediately evacuated to the United States for definitive diagnosis and therapy. This evacuation occurred six weeks after the initial incident. On presentation, weight bearing on the extremity was impossible. He was referred to a tertiary care center. The foot was extremely tender and swollen, and the extremity had a markedly decreased range of motion. The diagnosis of RSD was made. This was treated by placement of a lumbar epidural block. The result was total relief of pain, but he was still unable to move the extremity. It was decided to place a temporary epidural catheter and to infuse local anesthetic on a continuous basis. Over the next few days of intensive physical therapy, he remained pain free and had an approximately 90% return of function. The catheter was removed and improvement continued. Return of full function of the extremity occurred in two months, and he was returned to duty.

Discussion

The first description of RSD was by the military physician, Weir Mitchell, in 1864. He described Civil War casualties who suffered from "severe burning pain, the dystrophy of cutaneous tissue, glossy skin, and the atrophic muscle."[2] He was unable to treat these patients. Thirty years after the end of the war Doctor Mitchell's son was still caring for many of these patients.

During World War I, Leriche, a French physician, treated soldiers with RSD by periarterial surgical neurotomy (sympathectomy). This provided good relief of symptoms and a return of function. At this time, procaine, a local anesthetic, became available for clinical use. Leriche started using procaine to treat his RSD patients by producing a chemical sympathectomy.[3] He reported good results with this technique. Though many other treatment modalities have been studied, the chemical sympathectomy produced by local anesthetics remains the treatment of choice.

RSD can result from any number of insults, the most common of which is an accidental injury. Dislocations, sprains, fractures, traumatic amputations, crush injuries, and even minor cuts can trigger this syndrome. It can also be precipitated by iatrogenic means, for example: minor surgeries such as ganglion cyst removal, forceful manipulation, an excessively tight cast, or even medication injected in close proximity to a nerve. RSD has also been triggered by certain disease states such as herniated disc or diabetic neuropathy.

There is no correlation between the severity of the initial insult and either the onset or the severity of the resulting dystrophy. A minor injury is as likely as a major insult to cause RSD.

RSD is classically described in three phases. The first (acute) usually begins in days to weeks after the insult. The patient will often complain of a burning or aching pain, localized edema, tenderness, muscle spasm, hyperpathia, and hyperesthesia. The affected area will appear red and warm to the touch, and there will be accelerated hair growth. The pain is made worse with motion, activity, or sensory stimulus. The affected area will, however, be limited to the distal aspect of the extremity. If left untreated, the patient will remain in this phase for six weeks to six months.[6]

In the second phase (dystrophic), the pain is described as a constant burning or throbbing. The affected area will expand to include more of the limb. The skin appears cool, gray,

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and cyanotic. The edema will become brawny and tight, hair growth is markedly decreased, and nails brittle and cracked. Patients allow the nails to grow because they are too painful to cut. There will also be obvious muscle wasting, and a marked decrease in range of motion of the extremity.[6]

The third phase (atrophic) is the final stage of this condition, and most of the changes are permanent. The burning throbbing pain may be reduced because of a slightly increased blood flow to the extremity. The skin will become drawn and tight. There will be marked atrophy, and the patient will usually hold the extremity in a protected posture.[6]

The treatment of RSD hinges on early recognition of the process and an aggressive multidisciplinary treatment plan. First, the patient should have a series of chemical sympathectomies with local anesthetics. This will confirm the diagnosis, and totally relieve the pain.

For the upper extremity this would be either a stellate ganglion block or a cervical epidural. The ease of placement of the stellate ganglion block makes it the treatment of choice.[5] In the lower extremity the patient can have a lumbar epidural or a lumbar sympathectomy. The lumbar sympathetic blockade, though technically more difficult, is a more specific block. If there is a question of whether RSD exists, the lumbar sympathetic blockade should be performed.[5] A bretylium/guanethidine Bier block of an extremity can also be performed to provide a chemical sympathetectomy. All of these techniques are relatively easy to perform, but should be left to an anesthesiologist who is trained in pain management.

Regardless of which block is used, while the patient is pain-free he should be subjected to aggressive physical therapy to include range of motion and strengthening exercises. Physical therapy should continue to be performed while the patient is pain-free. The patient should then receive biofeedback training. If he can control the emotional triggers of an RSD flare-up, the recovery process will be much shorter. The acute phase of RSD will respond well to these therapies. As the patient enters the late dystrophic phase, and the atrophic phase, the chance of recovery dramatically decreases.

Reflex sympathetic dystrophy needs to be distinguished from causalgia, which is a syndrome of pain and autonomic dysfunction resulting from a major nerve trunk injury. The pain complaints of causalgia tend to appear out of proportion to the injury, and the affected area tends to defy normal dermatomal boundaries. The differential diagnosis of these disease states can be difficult, and is usually dependent on performing a sympathetic block. The pain of causalgia is afferent in nature, and a sympathetic blockade might block the efferent symptoms, but will not treat the cause of the pain. If a sympathetic blockade resolves the patients symptoms, the patient suffers from RSD; if not, the patient most likely suffers from a causalgia.

RSD has always been prevalent in military personnel. Most of the advances in the treatment of this disease have occurred in battlefield situations. The key to its management is early recognition and treatment. With the advent of local anesthetic sympathectomies, the duration of this disease has been greatly reduced. The health care provider involved with primary patient care is ideally suited to diagnose the disease and direct the patient to appropriate treatment. The case described above is a success story, the patient presented for medical therapy and was able to have the RSD stopped before it progressed to a permanent change. With the timely institution of aggressive therapy, the patient is able to quickly return to a functional lifestyle.

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