lower lobe viability. The upper lobe was resected due to its tumor burden. She continues to do well 1 year postoperatively. While we agree with a general approach of emergency surgery on diagnosis of pulmonary torsion, individual patients may demonstrate varied pathophysiologic responses to its occurrence.

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Reflex Sympathetic Dystrophy Following Arterial Blood Gas Sampling in the Intensive Care Setting*

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A 54-year-old woman developed signs and symptoms consistent with reflex sympathetic dystrophy in her left upper extremity following arterial puncture. Diagnosis was confirmed by bone scan, and sympathetic blockade with intravenous regional bretylium completely relieved her severe, intractable pain.

(CHEST 1995; 108:578-80)

** intravenous; RSD = reflex sympathetic dystrophy

Key words: arterial puncture; causalgia; reflex sympathetic dystrophy; sympathetic blockade

Reflex sympathetic dystrophy (RSD) is a form of sympathetically maintained pain that is characterized by constant burning, swelling, hyperesthesia, allodynia, and vasomotor changes that usually affects the extremities. The mechanism responsible for these signs and symptoms is thought to be an abnormal reflex mediated by the sympathetic nervous system, usually in response to trauma. Other predisposing factors might include surgery, myocardial ischemia and infarction, primary neurologic disorders, or infection. In approximately 10% of cases, no cause can be identified.1 Without early recognition and treatment, RSD can progress through three stages, resulting in intense pain and irreversible trophic changes. In this case report, we present a patient who developed symptoms consistent with reflex sympathetic dystrophy following arterial puncture.

** CASE REPORT

A 54-year-old woman was admitted to the intensive care unit (ICU) for respiratory failure that required endotracheal intubation and mechanical ventilation. Four days after admission to the ICU, the patient developed pain in her left wrist. Although she was intubated, she was able to communicate that her pain developed after a particularly painful and difficult arterial blood gas sampling. She described the pain as burning in nature. Her left arm, wrist, and hand were erythematous and edematous, as well as extremely tender to light touch, palpation, and motion. She was afebrile, and there was no elevation of her white blood cell count. Plain radiographs failed to show any bony abnormalities. A CT scan was also normal. A bone scan revealed increased activity and uptake in the phalangeal joints and metacarpal bones of the left hand (Fig 1). These clinical and radiographic findings prompted a diagnosis of RSD, and the pain service was consulted for management. Treatment of sympathetically mediated pain is based on sympathetic blockade. Inter-

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Common Signs and Symptoms of Reflex Sympathetic Dystrophy

<table>
<thead>
<tr>
<th>Stage 1 (Acute)</th>
<th>Stage 2 (Dystrophic)</th>
<th>Stage 3 (Atrophic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset after injury</td>
<td>Minutes to hours</td>
<td>2-3 mo</td>
</tr>
<tr>
<td>Edema</td>
<td>Soft</td>
<td>Brawny</td>
</tr>
<tr>
<td>Hair and nail growth</td>
<td>Increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Sweating</td>
<td>Variable</td>
<td>Increased</td>
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<tr>
<td>Temperature</td>
<td>Increased</td>
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<tr>
<td>Skin</td>
<td>Erythematous</td>
<td>Cyanotic</td>
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<tr>
<td>Pain</td>
<td>Hyperpathia/allodynia</td>
<td>Hyperpathia/allodynia</td>
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Rupture of sympathetic input will abolish the abnormal reflex and hopefully reverse this cycle. Options included stellate ganglion blockade, intravenous (IV) regional blockade with bretylum or guanethidine, or systemic treatment with phenoxybenzamine. Due to this patient’s anticoagulation and rather significant cardiac history, we chose sympathetic blockade, which was accomplished with bretylum administered IV after tourniquet exsanguination of the arm, followed by slow, intermittent release of pressure. Immediately, near complete relief of pain was noted, confirming a sympathetically mediated pain syndrome. Following the block, the patient was able to tolerate palpation and motion of the wrist and hand, so physical therapy was initiated. Her pain improved and no further intervention beyond physical therapy was needed.

**Discussion**

The diagnosis of RSD should be considered when there is the following: (1) a history of trauma, tissue damage, and/or infection; (2) constant burning or aching pain that is out of proportion to the extent of the injury, worsens with physical contact or use, and does not follow a radicular or peripheral nerve distribution; (3) vasomotor or sudomotor change; (4) trophic change; or (5) increased tracer uptake on bone scan.2,3 Since the symptoms of RSD are so nonspecific (swelling, pain, decreased range of motion, or hypersensitivity), care must be taken to rule out other diseases or conditions that may mimic RSD.2,4

When unrecognized, RSD may progress through three stages of development (Table 1). The acute changes often occur within hours of the initial injury, accompanied by increased perfusion, vasodilatation, and edema. Over the course of several months, a vasocostrictive phase ensues. Though pain continues unabated, edema becomes brawny and the extremity may become cyanotic. After months to years, chronic atrophy of skin and muscle may occur, along with contractures and a decrease in pain. At this stage, atrophic changes are usually irreversible.5

There is currently no single pathophysiologic hypothesis that completely explains sympathetic dystrophies adequately. Most theories postulate an abnormal feedback or reverberatory circuit at some level in the nervous system, which links cycles of peripheral afferent sensory input with efferent sympathetic hyperactivity. Cutaneous trauma or crush injury irritate peripheral sensory nerves, increasing afferent traffic to the spinal cord. Increased efferent motor and sympathetic outflow may then occur as a result of several postulated mechanisms: (1) Loss of myelin insulation around peripheral nerves may occur, causing a short circuit in nerve conduction peripherally and cross-stimulation and cycle formation, which might cause increased pain during periods of increased sympathetic activity, such as the emotional state of our patient in a critical care setting.6 (2) An abnormal state of heightened activity at the spinal cord level may exist, with sensitized neurons in the cord responding by sending increased painful information to higher centers, as well as increased sympathetic efferent impulses to the extremity, which further stimulate afferent sensory input to the cord, again creating a potential reverberating pathway.7,8 (3) Loss of normal inhibiting control over spontaneous sympathetic output at the cord level may occur, resulting from overstimulation of the central nervous system, again potentially explaining why emotional stress in a critically ill patient might influence sympathetic outflow and painful symptoms.9 Thus, it appears that both peripherally and centrally mediated factors are involved in the pathogenesis of RSD, suggesting that a variety of treatment modalities may be effective.

Although many treatments have been advocated for RSD, rational treatment is based on the theory that RSD is secondary to sympathetic mediation of pain, and that interruption of this sympathetic stimulation will abolish the reflex and the RSD cycle.5,10-16 In 1974, Hannington-Kiff17 reported the use of IV guanethidine as a treatment for causalgia, another frequently used name for RSD.13 Since then, other agents have been used in trials for sympathetic block-
ade, including reserpine, lidocaine, and bretylium.13-16 These studies have reported beneficial results in most patients for at least a short time. Guanethidine and reserpine are no longer available in the United States as IV preparations, making bretylium and lidocaine the more commonly used IV blocking drugs. Bretylium tosylate, a quaternary ammonium compound, is an adrenergic blocking agent with actions similar to guanethidine and reserpine. All three agents produce inhibition of responses to adrenergic stimulation by decreasing norepinephrine release at the neuromuscular junction.18,19 Lidocaine alone has not been satisfactory in relieving the pain of RSD, but when combined with bretylium, results are more effective and sustained.13,15 Some investigators combine lidocaine with bretylium to raise the threshold of tourniquet pain and to block burning pain on injection of bretylium should it occur.13

Bretylium proved to be an effective and safe agent in our patient. Although most patients require more than one treatment, our patient experienced an initial complete reversal of her pain and symptoms after only one block. Even though some pain returned, the improvement after one block allowed her to participate in physical therapy and with time, regain the use of her left arm.

Early recognition of RSD symptoms with a high index of suspicion should be maintained to ensure proper diagnosis of RSD. Weiss et al3 demonstrated the efficacy of using bone scans in the diagnosis of RSD. In this study, patients with normal scans had no current symptoms of RSD and did not develop any. Patients with abnormal scans either had symptoms of RSD at the time of the scan or developed symptoms within a 6-month period. The authors concluded that bone scans are useful diagnostic tools for predicting patients at risk for developing RSD.3 Indeed in our patient, an abnormal bone scan was the most important diagnostic test leading to the diagnosis of RSD.

Needle sticks for arterial blood sampling have not been reported in the literature as a precipitating cause of RSD. In the case of our patient, arterial blood sampling appears to have initiated the syndrome of RSD. Early recognition and treatment led to reversal of RSD and prevented the progression of this syndrome to its more severe and irreversible stages. To our knowledge, this is the first reported case of sympathetically mediated pain that developed after arterial puncture in the intensive care setting and was successfully treated. We might postulate that the procedure, in the setting of significantly heightened baseline sympathetic tone, combined to produce the syndrome. Interventional pain management can lessen the suffering of patients in the ICU who develop pain syndromes that can be treated with neural blockade.

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