Reflex Sympathetic Dystrophy of the Knee

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Abstract

Reflex sympathetic dystrophy (RSD) of the knee frequently does not present with the classic combination of signs and symptoms seen in the upper extremity. Pain out of proportion to the initial injury is the hallmark symptom. Symptom relief by sympathetic block is the current standard for confirmation of the diagnosis. Because invasive diagnostic procedures, such as arthroscopy, are likely to increase symptoms, evaluation with a noninvasive diagnostic modality, such as magnetic resonance imaging, is preferred. Generally, RSD should be treated before surgical intervention to correct any underlying intra-articular pathologic condition. However, surgery may sometimes be necessary before RSD symptoms resolve; in these cases, use of intra- and postoperative continuous epidural block can be successful. The initial treatment of RSD of short duration should be conservative; physical therapy modalities, including exercise and contrast baths, and nonsteroidal anti-inflammatory drugs are indicated. In the authors' experience, an indwelling epidural block using bupivacaine for several days followed by use of a narcotic agent, combined with functional rehabilitation, is the most effective management when noninvasive treatment has failed. Surgical sympathectomy can be successful, but should be reserved until repeated lumbar sympathetic block or more than one trial of inpatient epidural block has failed. Early diagnosis and early institution of treatment (prior to 6 months) are the most favorable prognostic indicators in the management of RSD.

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Schutzer and Gossling[1] define the syndrome of reflex sympathetic dystrophy (RSD) as an exaggerated response to injury of an extremity, manifested by four more or less constant characteristics: (1) intense or unduly prolonged pain, (2) vasomotor disturbances, (3) delayed functional recovery, and (4) various associated trophic changes. The clinical presentation varies, and the clinical course is difficult to predict. While some authors have described the syndrome as self-limited,[2,3] many have asserted that complete spontaneous resolution is rare.[4,5]

Bonica[6] (1973) was the first to use the term RSD to describe the syndrome of pain, decreased temperature, and abnormal searing sensations in an extremity. Historically, multiple terms have been used descriptively, including neurovascular dystrophy, posttraumatic vasomotor abnormality, traumatic angiospasm, sympathetic neurovascular dystrophy, postinfarctional sclerodactyly, causalgic state, minor causalgia, Sudeck's atrophy, minor traumatic dystrophy, shoulder-hand syndrome, major causalgia, major traumatic dystrophy, sympathetic-mediated pain, and pain dysfunction syndrome.[7]

Mitchell et al[8] (1864) initially used the term causalgia (Greek for "burning pain") to describe this syndrome. However, that term was classically reserved for pain syndromes following traumatic injuries to the major motor and sensory nerves of an extremity. Leriche[9] (1939) was the first to suggest the etiologic role of the sympathetic nervous system, and Sudeck[10] (1900) first recognized the association of RSD with regional osteoporosis. At present, RSD is the accepted terminology.[1,4]

Reflex sympathetic dystrophy is manifested by abnormal vasomotor, thermoregulatory, neurotrophic, sympathetic, and parasympathetic activity in the extremity.[1,3,4,11,12] It involves both peripheral and central nervous system abnormalities, and the involved extremity may become severely affected and dysfunctional. It is not a disease, but rather a pathologically exaggerated manifestation of a physiologic event. In the upper extremity, it has been widely recognized and extensively studied. In contrast, lower extremity involvement is less common and has a more varied presentation.[7] Reflex sympathetic dystrophy of the knee is even less well understood and has only recently been recognized.

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Review of the Literature

In 1977, Ficat and Hungerford[2] described their experience with RSD of the knee. They stressed that when the knee is the central area of involvement, the patellofemoral joint is always involved. In their experience, the most common inciting trauma was a direct blow to the patella. They considered the "vasomotor temperament" of the individual to be an important factor in precipitation of the syndrome. Vasomotor instability and intraosseous ischemia were suggested as possible exacerbating pathophysiologic mechanisms. They described three slightly different modes of onset: (1) The pain begins immediately after the inciting trauma or surgical intervention, and is out of proportion to the inciting trauma. (2) The postinjury or postoperative course is normal, but the predicted recovery does not occur. Instead, the pain continues or even increases. Mobilization of the knee becomes difficult. (3) The postinjury course is as expected, and the patient makes a good recovery and may even become symptom free. The pain then reappears, perhaps in response to overvigorous or inappropriate physical therapy. The RSD syndrome then becomes manifest.

Ficat and Hungerford[2] proposed a protocol of medical management including physical therapy and sympathetic block. Surgical sympathectomy and core decompression of the patella were used in selected cases. Significant improvement was obtained in 80% of 15 patients who underwent core decompression of the patella, but that procedure has not been reported as useful by other investigators.

Reider and Rattenborg[13] (1985) reported four cases of RSD of the knee treated with chemical lumbar sympathectomy under computed tomographic control. They found this an excellent diagnostic and therapeutic technique in patients who have not responded to more conservative treatment measures.

Tietjen[5] (1986) reported the cases of 67 patients with unexplained knee pain, 14 of whom met the criteria for a diagnosis of RSD. Many of these 14 patients had an associated compensation or liability claim. He described three stages of RSD: (1) early, in which pain is the presenting symptom; (2) dystrophic, in which the classic discoloration and skin temperature changes are present; and (3) atrophic, in which muscle atrophy and joint changes have occurred. Typical radiographic findings were present within 2 to 4 weeks of the onset of symptoms, with osteoporosis of the patella being most frequent. Bone scans demonstrated increased uptake in two thirds of patients, but the arthrographic and arthroscopic findings were normal.

His treatment protocol included nonsteroidal anti-inflammatory drugs (NSAIDs) and oral corticosteroid preparations. Avoiding narcotic medications was emphasized. The use of alternating warm and cool whirlpools seemed beneficial. Although bracing did not appear to be useful, an elastic compressive bandage was of some benefit.

Ogilvie-Harris and Roscoe[14] (1987) reported 19 cases of RSD of the knee. Their patients were treated with NSAIDs, analgesics, physical therapy, and sympathetic blocks. Epidural morphine was used in selected patients. When patients were treated within 6 months of the onset of symptoms, over 70% achieved an excellent result. Of those treated later, none achieved an excellent result, and only 22% had a good result. At follow-up averaging 3.4 years, no patient had completely recovered, on the basis of objective testing results.

Katz and Hungerford[15] (1987) reported an additional 36 cases of RSD of the knee. Injury to or operation on the patellofemoral joint triggered the syndrome in 64% of their patients. Coexistent internal derangement of the knee was present in 64% of patients. Their sine qua non diagnostic test, as well as the mainstay of their treatment, was lumbar sympathetic block. Physical therapy, analgesics, and sympatholytic pharmacologic agents were also employed. Most of their patients had long-standing, severe involvement. When sympathetic block or sympathectomy was performed within 1 year of the onset of symptoms, patients had a significantly better recovery as measured by pain and function scores. The authors concluded that early diagnosis and treatment were the keys to successful management.

Cooper et al[7] (1989) reviewed the data on 14 patients with RSD of the knee. Pain out of proportion to the severity of the injury was present in all 14 patients. However, variation in clinical severity was characteristic of the presentation. The diagnosis was confirmed if symptoms were relieved by lumbar sympathetic block. All 14 patients had extensive physical therapy and medical treatment before continuous epidural sympathetic block was administered by means of an indwelling catheter for an average of 4 days. The average length of follow-up was 32 months. Eleven patients had complete resolution of the symptoms, two patients had sufficient intermittent aching with changes in the weather to require medication, and one patient had no relief.

Ladd et al[16] (1989) reported the cases of 11 patients with so-called sympathetic imbalance of the knee treated by an epidural block protocol similar to that proposed by Cooper et al.[7] All but one patient had an initial favorable response. How-

ever, five patients required readministration of the block because of a clinical relapse. Like others, Ladd et al observed that recovery was typically prolonged, particularly when the diagnosis was delayed.

Finsterbush et al[17] (1991) reported the cases of 18 patients with RSD of the knee treated by epidural block. Patellofemoral joint involvement was universal. Initial misdiagnosis of the syndrome led to numerous unsuccessful surgical procedures in six patients, and three patients had undergone knee fusion. Twelve patients had worker's compensation claims. Early diagnosis and treatment were stressed as the keys to successful management.

O'Brien et al[18] (1991) reported RSD of the knee confirmed by diagnostic sympathetic block in 60 adult patients. Pain out of proportion to the trauma and vasomotor changes, including mottling of the skin and temperature changes, were reliable in predicting a positive response to sympathetic block. Bone scanning was less reliable. Using multiple repeated sympathetic blocks (an average of nine), they obtained good results in 92% of their patients. In contrast to previous reports, the length of time from initial injury and the number of blocks required were not significant prognostic indicators. Interestingly, their protocol employed the use of multiple blocks over a relatively short period of time rather than the use of a continuous epidural block. In more than half (66%) of their patients, RSD developed after surgery, which was arthroscopic in 30% of the cases. The results of treatment were directly related to the presence of anatomic pathology. The results were much more favorable in knees with either no identifiable lesion or a surgically correctable lesion than in those with an uncorrectable lesion.

Wilder et al[19] recently reported a series of cases of RSD in children. The lower extremity was affected in 87% of their cases, approximately one third of which involved the knee. They emphasized the variety of symptoms encountered in young patients and the need for a multidisciplinary team approach to case management. Sympathetic blocks were used selectively after failure of more conservative measures. Wilder et al were the first to emphasize that the diagnosis of RSD cannot and should not be completely excluded even after a negative response to a confirmed sympathetic block.

Reflex sympathetic dystrophy as a complication of total knee arthroplasty has been only rarely reported.[15,20] Cadambi and Jones[20] recently reported 14 cases in which RSD developed after total knee arthroplasty. These 14 cases and the five reported by Katz and Hungerford[15] are the only reported cases of RSD after total knee replacement.

Epidemiology

Reflex sympathetic dystrophy of the knee is more common in adults than in children. Of the 224 cases of RSD of the knee noted in a literature review, 70% occurred in female patients, who had an average age of 38 years. The syndrome is rare in black persons.[16,18] Patients may have a predisposing diathesis or may be sympathetic "hyperreactors," as evidenced by a history of increased sweating in the palms and poor tolerance of cold, and are often described as emotionally labile. Whether or not these physiologic and psychological factors predispose a patient to RSD, they certainly are known to aggravate the syndrome, making management more difficult. Because of this underlying diathesis, caution should be exercised when considering surgical intervention in any patient with a history suggestive of RSD. This caution should be balanced against awareness of the importance of surgical correction of any well-defined pathologic condition that may be triggering the syndrome.[18]

Pathophysiology

Several excellent detailed reviews of the pathophysiology of RSD are available,[1,3,6,11,12,21,22] which will be summarized here.

The usual response to trauma includes a normal degree of sympathetic discharge accompanying pain followed by subsequent symptom resolution. The abnormalities leading to an exaggerated sympathetic nervous system response are poorly understood. Abnormal prolongation of sympathetic discharge or failure to disrupt the process because of continuing trauma permits the underlying symptoms to escalate. If untreated, this process leads to RSD, which continues until there is permanent dysfunction of the extremity.

Several theories have been formulated to attempt to explain the etiology of this disorder. Livingston[21] (1943) suggested that the vicious circle is initiated by somatic pain that leads to excessive sympathetic discharge. Melzack and Wall[22] (1965) advocated the "gate control" theory of pain interpretation by the central nervous system. They described special cells in the substantia gelatinosa of the dorsal horn of the spinal cord that modulate the transmission of afferent impulses from peripheral sensory nerves. They suggested that these special cells interpret the sensory impulses and relay them to the brain as messages of pain. Impulses transmitted on large, myelinated afferent fibers "close the gate to pain," but impulses transmitted along the small C fibers "open the gate," thus allowing a small stimulus to be perceived as a great deal of pain. The exact cause of ongoing

sympathetic discharge, however, is poorly understood.

Stages

Classically, RSD has been characterized as occurring in three stages.[1,3,4] The first stage consists of swelling with edema and increased temperature in the extremity. An exaggerated pain response is present, accompanied by apprehension of any range of motion of the affected joint. Hyperhidrosis is common, and allodynia is a frequent manifestation.

After approximately 3 months, the initial edema becomes more brawny, a characteristic of the second stage. Hyperhidrosis may extend into the second stage. Trophic changes of the skin begin to appear, and the region may become engorged and cyanotic. Joint motion continues to decrease.

The third stage usually begins 6 to 9 months after the onset of symptoms and may last for years. Although the pain may diminish in degree, it may continue for many years. Trophic changes are more pronounced, edema is less prominent, and the skin becomes paler, cooler, and drier. Thinning of the skin and subcutaneous tissues develops, producing a glossy appearance. Joint stiffness predominates and may become permanent.

It is important to remember that these classic stages are more typical of RSD of the hand and may not be present in RSD of the knee. Therefore, it is mandatory that a high index of suspicion be maintained when evaluating any patient with knee pain that is out of proportion to the inciting trauma or surgery.

Signs and Symptoms

Since the best treatment is prevention by early mobilization and rapid progression to a functional gait pattern, early diagnosis is the key to successful management. Pain out of proportion to the severity of the initial injury is the hallmark symptom and should alert the clinician to the possible diagnosis of RSD. Although patients may exhibit severe patellofemoral symptoms, early loss of motion is an important feature in distinguishing RSD of the knee from other patellofemoral arthralgias.[2,15] It has been our experience that loss of flexion is a more common finding than loss of extension.[7]

The classic signs are atrophic skin changes, decreased temperature, hypersensitivity to touch, swelling, and increased sweating. The pain is described as burning, searing, aching, or boring and is nondermatomal in distribution. There is the potential for both vasodilatory and vasoconstrictive signs. Vasodilatation due to *decreased* sympathetic activity produces warm, flushed, dry or scaly skin. Vasoconstriction due to *increased* sympathetic activity produces cool cyanotic or pale skin, which tends to be moist. In the early stages, the subcutaneous tissue may be edematous. In the later stages, the subcutaneous tissue is firm and atrophic. As the disorder progresses, joint stiffness becomes a more predominant finding.

Patients with RSD of the knee often do not have this classic combination of signs and symptoms or the temporal progression of distinct stages. Instead, there is a marked variability in the clinical syndrome.[4]

When the knee is the central area of involvement, the patellofemoral joint is always affected.[2,7,17] The patellofemoral signs are varied and may include retinacular induration and tenderness, decreased patellar mobility with hypersensitivity to palpation, and patellar tenderness. The presence of an effusion is not common.[5]

Diagnostic Evaluation

Osteopenia of the patella is the most common radiographic finding; however, it may take some time to appear. The more diffuse osteopenia of classic Sudeck's atrophy is less commonly seen in the knee.

Although bone scans and thermograms may be abnormal, they are not specific to the diagnosis of RSD and are not considered to be essential. Some authors suggest diagnostic arthroscopy to exclude "triggering" intra-articular pathology; however, magnetic resonance (MR) imaging will provide the same information noninvasively. This is a major advancement, since surgical intervention has been clearly demonstrated to be a precipitating cause of the onset and exacerbation of RSD.[7]

The most reliable diagnostic test is symptom relief by successful sympathetic block. The diagnosis of RSD is considered firm if the pain is significantly improved for the duration of action of the anesthetic agent used for the block. The sympathetic block is judged successful if it is followed by a documented increase in the temperature of the skin of 1° C.

Although a successful sympathetic block remains key to diagnosis, recent reports suggest that some lumbar sympathetic fibers may bypass the sympathetic chain and therefore not be blocked by classic lumbar sympathetic block.[23] Therefore, it is possible that a successful sympathetic block might not provide pain relief for the patient with RSD of the knee. While failure to respond to a diagnostic sympathetic block should not be strict grounds for excluding the diagnosis of RSD in the lower extremity, a completely negative response to confirmed lumbar sympathetic block should still suggest a diagnosis other than RSD of the knee.[19]

The recent increase in awareness of RSD has led to the tendency to use it as a catchall diagnosis for patients with unexplained anterior knee pain. Although we are in agreement that certain patients with RSD may not demonstrate a positive response to a sympathetic block, this is a rare occurrence. The clinical setting should dictate the course of action. If the symptoms strongly suggest RSD and the diagnostic block is negative, the diagnosis should not be strictly excluded. However, if the signs and symptoms do not suggest RSD and the diagnostic block is negative, the diagnosis of RSD should not be rendered simply because the pain is difficult to explain.

Treatment

According to the literature, the initial treatment of RSD should include gentle exercise, the avoidance of aggressive manipulation, massage, contrast baths, biofeedback, limb elevation to control edema, NSAIDs, antidepressive medications, and psychological evaluation. Systemic corticosteroids and propranolol have also been reported to be useful.[24-27]

Failure to respond to these noninvasive modes of treatment should be followed by the use of sympathetic blocks, which have become the standard of care in difficult cases.[28] A variety of techniques have been employed. Laurin[29] recommends regional blocks using lignocaine, and Poplawski et al[3] recommend regional blocks using lidocaine (Xylocaine; Astra) and methylprednisolone sodium succinate (Solu-Medrol; Upjohn). Reider and Rattenborg[13] report excellent results with chemical lumbar sympathectomy using bupivacaine or alcohol. Guanethidine[30,31] or reserpine and guanethidine[32] given intravenously can produce chemical sympathectomy for as long as 3 or 4 days, during which time physical therapy may be employed. Lankford and Thompson[4] recommend surgical sympathectomy in patients who have undergone four sympathetic blocks without complete relief of symptoms, while others have been more aggressive in the number of blocks used.[18] Our algorithm is based on the use of more aggressive epidural blocks.

In addition to sympathectomy, other adjunctive treatments have been suggested. Ficat and Hungerford[2] achieved variable results with core patellar decompression but did not recommend it as the sole treatment. An important consideration in the patient with well-established RSD of the knee is whether it continues to be exacerbated by correctable intra-articular pathology. This can be excluded by MR imaging without the drawbacks of surgical exploration.

We have established a treatment algorithm based on our experience[7] (Fig. 1). In patients with suggestive clinical signs and symptoms of less than 6 weeks' duration, we initiate a trial of an NSAID, intensive but pain-free physical therapy (to increase motion and increase strength), alternating hot and cold soaks, and progressive weight-bearing. Patients who respond to this initial treatment usually progress to resolution of their symptoms. If there is no relief of symptoms or if the symptoms of RSD have been present for more than 6 weeks and are progressing in severity, we proceed directly to a diagnostic sympathetic block.

The sympathetic block serves two purposes. First, if it relieves the symptoms during the duration of action of the local anesthetic used, the diagnosis is confirmed. Second, a single block may terminate the symptoms.

If the symptoms recur after the block, in-hospital treatment with an indwelling epidural catheter is undertaken. On the basis of our reported experience, we believe that early intervention with a continuous indwelling epidural block is the most successful form of treatment for the patient with established RSD (duration of more than 6 weeks) and for the patient who does not respond to outpatient sympathetic blocks.

The epidural block has several advantages over a standard sympathetic block. While a sympathetic block may relieve pain resulting from sympathetic hyperactivity, it provides no relief of somatic pain. Therefore, if a stiff joint is aggressively mobilized after sympathetic block, the pain may recur and the pain cycle may be restarted. Epidural block allows pain-free joint mobilization because it blocks both sympathetic pain fibers and somatic pain fibers. An epidural block is easy to perform, and a lumbar epidural catheter may be left in place for up to 7 days.[7] This technique allows manipulation of the knee as needed and the use of continuous passive motion, both of which increase range of motion. Also, an indwelling continuous-drip epidural block eliminates the need for repeated sympathetic blocks, which can be difficult to perform and painful.

Another advantage is that various medications can be used to meet individual patient needs during the course of treatment. Initially, bupivacaine provides a sympathetic, sensory, and motor block that is excellent for permitting increased range of motion without pain. The initial dose of bupivacaine is 1 mg of a 0.5% solution per kilogram of body weight. After this is administered, the continuous drip is set at 0.25 to 0.5 mg/kg per hour and is titrated to give complete epidural anesthesia. If stiffness is a problem, manipulation may be performed early under this epidural block. We consider an arc of flexion of less than 90 degrees an indication for manipulation.

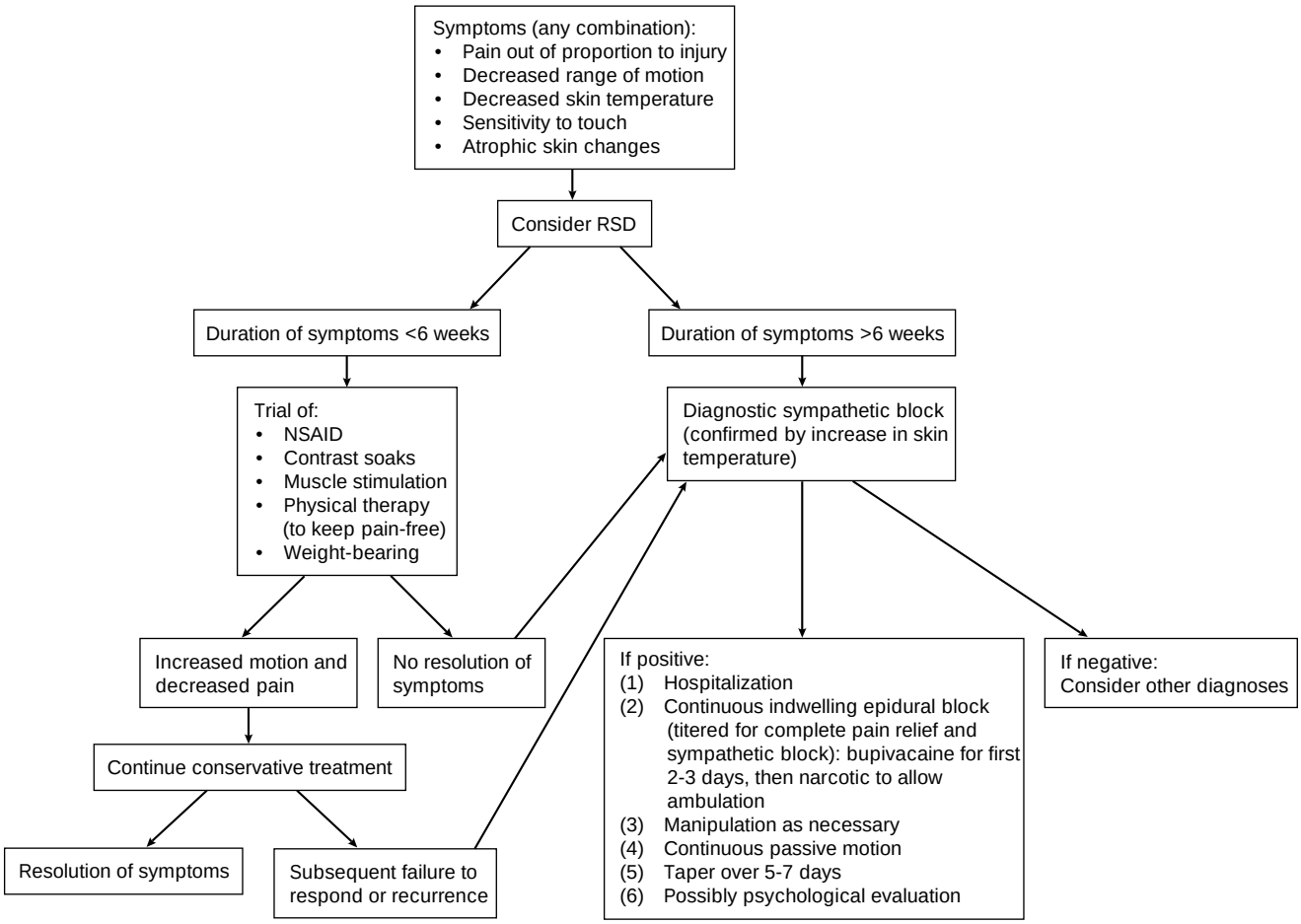

Fig. 1 Treatment algorithm for RSD of the knee.

Later in treatment, a narcotic epidural agent (morphine, demerol, or fentanyl) can be administered to provide pain relief without producing a complete motor block. This enables the patient to be ambulatory and to bear weight on the limb during the sympathetic block. However, narcotic epidural agents are used only after the patient has regained pain-free motion with physical therapy and continuous passive motion.

These narcotics have advantages and disadvantages, and the selection of the specific agent is left to the discretion of the anesthesiologist specializing in pain management. The switch from bupivacaine to a narcotic is instituted empirically, usually after 2 to 3 days. Morphine is given as an intermittent bolus of 0.07 mg/kg every 10 to 18 hours. Demerol is given as a bolus of 1.0 mg/kg followed by continuous infusion of 0.1 mg/kg per hour. Fentanyl is given as a bolus of 1.0 μg/kg, followed by infusion of 0.3 μg/kg per hour. Because of its potential for respiratory depression, morphine is not usually infused continuously. The continuous infusion of demerol or fentanyl provides consistent analgesia. The epidural block is tapered over 5 to 7 days.

Our experience suggests that approximately 80% of patients experience a dramatic initial response to this treatment protocol and seem to obtain more lasting relief of their symptoms than they would following a simple diagnostic sympathetic block.[7] If the patient subsequently ceases to respond or has a recurrence of symptoms, this inpatient protocol may be repeated. Multiple repeated lumbar epidural blocks are not usually necessary in these patients, as is frequently the case with lumbar sympathetic blocks. However, repeating the inpatient epidural protocol once or twice should be tried before considering surgical sympathectomy. The use of chemolytic agents, such as phenol, to effect chemical sympathectomy can be successful, but this involves a greater risk of complications and is

more controversial. Development of anesthetic agents with a very long duration of action (months) will be of great value in repeated sympathetic block for RSD.

Special Considerations

Arthroscopic Procedures

Reflex sympathetic dystrophy seems to be commonly associated with arthroscopic surgical procedures.[7] Whether these procedures exacerbate preexisting unrecognized RSD of the knee or are the inciting event is difficult to determine retrospectively. Tietjen[5] reported that 3 of his 14 patients had undergone arthroscopy and 5 had undergone arthrotomy. He did not mention whether these procedures had preceded the onset of symptoms, but he believed that the patients who had undergone arthrotomy improved more slowly than the others. Ogilvie-Harris and Roscoe[14] performed diagnostic arthroscopy on all of their patients after the diagnosis of RSD was made. They believed it was important to rule out serious intra-articular pathologic conditions, as many of their patients seemed to have a locked knee at the time of initial examination. However, no serious intra-articular lesions were found in their patients.

In our report,[7] 11 of 14 patients had undergone a patellar operation before the diagnosis of RSD was made. However, in 9 of the 11 the history suggested that RSD was present before the operation. Therefore, one should look for symptoms of RSD before considering surgical treatment of the knee. The frequent association of previous arthroscopy of the knee with a confirmed diagnosis of RSD suggests that the "look and see" philosophy of evaluating knee pain is rarely justified. Today, MR imaging can exclude the presence of significant mechanical problems in the knee and thereby avoid the potential exacerbation of RSD symptoms by ill-advised arthroscopy.[33,34]

If MR imaging confirms the presence of a significant mechanical cause of pain in the patient with RSD, therapeutic (not diagnostic) arthroscopy can be performed with the use of epidural anesthesia, which can be continued postoperatively via an indwelling catheter as outlined above.

Multiple Operations

A particularly difficult situation arises in patients who have had multiple previous surgical procedures and who have constant pain. While some of these patients have intra-articular pathology, usually related to arthritic changes, it is important to recognize that they may also have extra-articular soft-tissue pain caused by multiple factors. Tender scar formation, scar adherence to underlying structures, and neuroma formation (especially of the saphenous nerve branches) may be contributing factors. These patients may also have increased sympathetic activity suggestive of RSD. The treatment of patients with somatic pain with a confirmed anatomic basis and RSD is very difficult and must be individualized.

Great caution must be exercised in recommending total knee arthroplasty, especially for the younger patient with severe knee pain after multiple failed ligament or soft-tissue procedures. While it may be tempting to recommend total knee arthroplasty as a salvage procedure, the results are often tragic. Likewise, knee arthrodesis may not produce a pain-free extremity.

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