Probable Reflex Sympathetic Dystrophy of the Penis

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(Received 27 August 1985, revised received 20 November 1985, accepted 26 November 1985)

Introduction

Since Mitchell's classic description of brachial plexus causalgia resulting from gunshot wounds during the Civil War [6], numerous articles have appeared that have added to the clinical labels for syndromes relating to dysfunction of the sympathetic nervous system. A number of terms, including reflex sympathetic dystrophy (RSD), minor causalgia, major causalgia, and Sudeck's atrophy have been generically applied, and various parts of the body have been shown to be affected. We are not aware of a previous report in which the penis has been involved by RSD, but the following illustrates such a case.

Case report

A 57-year-old, previously healthy male presented to the Pain Management Center with a 6-week history of disabling penile pain. The pain was described as being intensely burning in quality and creating considerable social embarrassment because the patient could not tolerate any intentional or accidental touch stimulation to his penis. Thus, he developed a remarkable degree of avoidance behavior. Participation in intercourse was impossible because of the pain, though he reported no difficulty in achieving erection. Urination was not inhibited.

The patient's past medical and surgical history revealed only that he had undergone an uncomplicated transurethral resection of the prostate (TURP) 1 year previously. He took no medications, was a non-smoker, and had no psychiatric history.

Upon clinical examination, the penis was extremely sensitive to touch. Discoloration, altered hair patterns, swelling and other obvious stigmata of RSD were not apparent. The temperature of the shaft away from major vascular structures was
28°C. A psychological evaluation was obtained in the Pain Center to supplement the assessment of the patient, and no marked abnormalities were discovered.

Because of the description of the pain as burning and the relatively decreased temperature, a caudal epidural block was performed empirically. Using standard aseptic technique, 20 ml of 1% plain lidocaine were injected in the epidural space. After 15 min, the penile temperature had increased 5°C and the shaft blood flow as measured by laser doppler technology (Med-Pacific) had increased 200%. The patient obtained approximately 1 h of total pain relief. Encouraged that the patient's pain might represent an early RSD and be responsive to repeated blocks, a total of 3 caudal epidurals was performed (on a one-per-week basis) with 20 ml of 0.25% plain bupivacaine. After the first block with bupivacaine, the patient reported 2 days of pain relief. The second block resulted in 6 days of symptomatic relief; and after the third caudal block with bupivacaine, the patient had no burning pain for 6 months. The patient reported no sexual dysfunction at any time during or after the therapy with the nerve blocks.

Discussion

The penis has a well-defined sympathetic nervous system supply. The postganglionic outflow originates from the hypogastric and pelvic plexuses. Likewise, the prostate is innervated from these same sources. We hypothesize that this patient received an injury to the sympathetic nervous system as a result of his TURP. The apparent delay in the onset of symptoms in this case is not unusual. RSD is a dynamic clinical syndrome that may become symptomatic days to months after the original trauma, such that some patients can make no temporal association of any trauma to the onset of their pain.

Another possible explanation for the patient's complaints could be the presence of an underlying psychological problem. Our patient was evaluated with the SCL-90 (symptom check list), our in-depth pain-psychosocial questionnaire (“Pain Assessment Inventory and Narrative”), an interview with a physician, and an interview with a psychologist. The patient's spouse was interviewed by the physician and psychologist. The patient subsequently was seen in follow-up on 3 occasions, and no obvious psychological abnormalities were noted at any time.

There is no single, all-encompassing, pathophysiologic theory to explain RSD. The most widely held theories revolve around the creation of abnormal reverberatory circuits at some level in the nervous system that contribute to a vicious circle of afferent sensory input and efferent sympathetic hyperactivity. Doupe and his colleagues proposed that there was a loss of myelin insulation around neural structures in the periphery that resulted in a short circuit [3]. Livingston postulated that some kinds of injuries triggered abnormal reverberatory activity in the internuncial neurons in the spinal cord [4]. Melzack, the co-originator of the Gate Control Theory, claimed that damage to the afferent input to the brain-stem reticular formation (which acts as a ‘central biasing mechanism’) resulted in loss of normal inhibitory control over spontaneous sympathetic output [5].
Therapeutically, interruption of the sympathetic outflow produces excellent results in RSD, especially when treatment is instituted early, and before atrophic changes occur [1,2]. In this case, the sympathetic outflow was successfully interrupted with caudal epidural techniques using local anesthetic drugs. We will be interested to see if additional similar cases are reported in men following transurethral prostatic resection. We think this patient had a rather typical early RSD that responded to appropriate treatment. Because the location of RSD in the penis does not seem to have been described previously, we suggest that RSD should be included in the differential diagnosis for men who have complaints of penile pain. Though our patient had recently undergone urologic surgery, one wonders if non-surgical trauma to the penis could also precipitate RSD-like symptoms.

References