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Causalgia of Vascular Etiology Following an Abdominal Injury

MARCOS SZEINFELD, M.D.,* ROBERTO SAUCEDO, M.D.,† VICENTE S. PALLARES, M.D.‡

Causalgia is a pain syndrome characterized by chronic, severe, constant, burning pain which frequently is associated with vasomotor disturbances, delayed return of function, and trophic changes.[1] Major causalgia is often seen following penetration of the body by a high-velocity projectile, near or through peripheral nerves in the upper or lower extremity.

An unusual case of severe causalgia affecting the lower extremity secondary to a gunshot wound to the abdomen is described. The uniqueness of this case was the early development of symptoms of causaligia following vascular abdominal trauma with complete absence of peripheral nerve injury.

Report of a Case

A 21-year-old, healthy man was admitted for a gunshot wound to the abdomen. A 38-caliber bullet entered the right upper quadrant and traveled in a downward direction from right to left and front to back. The exit of the wound was located 1 cm above the left iliac crest at the level of the posterior axillary line.

The patient arrived at the Emergency Room in critical condition and underwent an emergency exploratory laparotomy which included resection of the third and fourth portion of the duodenum, repair of the superior mesenteric vein, and repair of an extensive injury to the aorta below the renal arteries. After surgery, the patient was transferred to the Surgical Intensive Care Unit in stable condition. The postoperative course was uneventful until the fifth day, when he began to complain of burning pain in the left buttock and left leg. Within five days, the pain became unbearable and could not be relieved completely with narcotic analgesics. Four weeks after the accident, the patient was referred to the Pain Clinic for evaluation of the persistent pain. Prior to our consultation, a pseudoaneurysm of the abdominal aorta was ruled out.

When the patient was first seen, the pain was severe, and he would not allow anyone to touch the affected part. The pain was constant and characterized as pulsatile and burning in nature, radiating from the left buttock toward the posterior aspect of the left thigh and calf through the plantar aspect of the foot. The pain was exacerbated with heat and when lying in the right lateral decubitus, but some relief was obtained in the left lateral decubitus position.

On physical examination he was uncooperative, irritable, and extremely anxious. The skin of the left leg was cold and moist with no atrophic changes. The neurlogical examinations before and after the treatment did not show any sensory or motor deficits. The rest of the examination was within normal limits. A diagnosis of causalgia was made, and a diagnostic left lumbar sympathetic nerve block was carried out the same day. A 22-gauge needle was inserted 5 cm lateral to the L2 spine, and 15 ml of bupivacaine 0.25 per cent was injected. The pain disappeared almost completely within a few minutes after the block, and the extremity became warm and dry. There was an average increase of 5° C in the skin temperature in the left toes. The effect of the block lasted for over 10 hours. This procedure was repeated twice with the same results. As symptoms recurred after each block dissipated, and as the intervals between each block did not increase, the patient was advised to have a left lumbar sympathectomy to obtain

* Assistant Professor of Anesthesiology.
† Fellow of Anesthesiology.
‡ Professor of Anesthesiology.

Received from the Department of Anesthesiology and Pain Clinic, University of Miami School of Medicine, P. O. Box 016370-R-370, Miami, Florida 33101. Accepted for publication January 13, 1982.

Address reprints request to Dr. Marcos Szeinfeld.

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permanent pain relief. At this time the patient's spirits had improved markedly, and he had become very cooperative and optimistic.

One week later, the patient underwent an extensive left lumbar sympathectomy. After surgery, the patient was pain-free. A follow-up six months later revealed that the pain had not recurred since surgery. The patient was able to return to his normal activities.

Discussion

The development of causalgia following a gunshot wound to the abdomen with lesion to the abdominal aorta but without somatic nerve lesion has not been reported. Usually the cases of causalgia reported are injury or trauma to the brachial plexus, median and ulnar nerves in the upper extremity, and sciatic nerve or its terminal branches in the lower extremity.[2-6] The incidence of major causalgia following peripheral nerve lesion is approximately 2–5 per cent; the upper extremity being involved more often than the lower one. In the majority of cases reported, the lesion of the nerve was incomplete; only a few cases were reported of complete nerve section.[5] In this case the high-velocity projectile was distant from the sciatic nerve or the lumbosacral plexus. There was no clinical evidence of nerve damage.

The high-velocity projectile caused extensive damage to the aorta below the renal arteries at the level of L3 but did not produce any damage to the lumbosacral or sciatic nerve. The mechanism of causalgia in our case probably was initiated by damage to the perioaortic sympathetic plexus. The trauma produced an instability of the sympathetic nervous system which led to increased activity of the plexus. This possibly created abnormal pathways which were transmitted centrally and triggered intense activity in the internuncial neurons of the spinal cord. The internuncial pool acts as a receiving station which determines the routing of sensory impulses. These neurons synapse with the preganglionic sympathetic fibers, setting up an increased sympathetic efferent flow. The sympathetic impulse traveling down the efferent nerve will produce depolarization of the adjacent somatic sensory afferent nerve. The repetitive depolarization may create an increased firing of afferent discharges into the sympathetic pool of neurons in the spinal cord. The activities of the internuncial pool are disturbed, resulting in a state of imbalance and spreading of impulses upwards, downwards and to the periphery. This explanation allows for continued reflex sympathetic activity without the occurrence of "artifical synapses" at the site of injury.[7,8]

At the present time, sympathetic interruption is the treatment of choice. The analgesic effect of a sympathetic block is so constant that relief of pain following a sympathetic block should be considered an important diagnostic feature of causalgia. In fact, if the patient's pain is not temporarily relieved following a sympathetic block, the diagnosis of causalgia is in doubt. Sympathetic interruption can be accomplished clinically with a regional intravenous block, with guanethidine or reserpine, or by blocking the sympathetic chain with neurolytic agents such as phenol or alcohol. In our opinion, the treatment of choice for major causalgia in healthy patients, after a trial with long-lasting local anesthetics, should be a surgical sympathectomy. A surgical sympathectomy properly done produces excellent results with complete and long-lasting relief of pain. Statistics show that only 4 per cent of the cases had poor results.[4-5] The failures observed were probably secondary to incomplete sympathetic denervation due to anatomic variation in the abdominal sympathetic chain. On the other hand, chemical sympathectomy offers the advantage that it is a simpler procedure, but less predictable.

Physicians dealing with acute trauma cases should be aware of the possibility of developing causalgia following exclusively vascular abdominal trauma. Early recognition and aggressive treatment of causalgia favor an optimal clinical outcome, since spontaneous remissions are rare. If relief from repeated sympathetic blocks becomes less effective or static, and the initial response is dramatic but of short duration, surgical sympathectomy should be considered.[9] Early sympathectomy should prevent the occurrence of irreversible trophic changes as well as obviate the establishment of fixed pain patterns, secondary gains, and narcotic addiction, making the functional rehabilitation of the patient difficult or impossible.

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