Thoracic Lateral Cutaneous Nerve Entrapment Syndrome Without Previous Lower Abdominal Surgery

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Nerve compression, entrapment, or irritation syndromes are widely distributed, both anatomically and geographically. From Morton's neuroma at the base of the third toe[1] to greater occipital neuralgia of the scalp,[2] these syndromes often lead to diagnostic confusion, unnecessarily aggressive investigation and treatment, anxiety, and the unfair labeling of certain unfortunates as "somaticizers." Criteria for diagnosis usually include (1) an ability to reproduce the characteristic pain either by a maneuver on the part of the examiner or a particular motion by the patient, (2) noncontributory ancillary investigation, and (3) an unequivocal, favorable response to nerve block.[3] On physical examination, the involved area may be tender and hyperalgesic, and after a time, the patient may come to complain of a pain that is constant rather than dependent on a well-defined movement or posture.

At least three nerve entrapment syndromes of the abdominal wall have been reported, all in connection with antecedent surgery.[4] Described herein is a patient with lower abdominal pain and a history of trauma who responded to an infiltration block of the lateral cutaneous branches of the 10th and 11th thoracic nerves.

CASE REPORT

A 36-year-old housewife complained of severe pain in the left lower abdomen of 3 years' duration. It was intensified by lifting objects weighing more than 3 kg and by exercise. The patient described the pain as dull and superficial, and she had noted paresthesia in the affected area. She attributed the problem to an abrupt movement she had made during the 7th month of her last pregnancy, when she experienced an excruciating pain in the left upper abdomen, much as though a muscle were torn. There was no temporal association between her complaint and eating or bowel habits, and she had no symptoms that could be ascribed to the urogenital tract.

There were no findings on physical examination other than slight tenderness accompanied by hypoesthesia over the area of which she complained. Findings on abdominal ultrasonography and upper gastrointestinal x-ray films were negative. The possibility of a nerve-entrapment syndrome was considered despite the absence of a history of abdominal surgery, and a selective block of the lateral cutaneous branches of the 10th and 11th thoracic nerves on the left was performed as follows: A standard 8-cm, 25-gauge spinal needle attached to a syringe containing 2 mL of 0.5% bupivacaine, 1 mL of 2% lidocaine hydrochloride and 2 mL (3 mg) of betamethasone sodium phosphate and betamethasone acetate was introduced subcutaneously just superficial to the external oblique muscle and redirected in three passes in such a manner that a fan-shaped deposition of the anesthetic was accomplished[4] (Figures 1 and 2). For 3 days after the procedure the patient continued to complain of pain, but on the 4th day it ceased abruptly, and she was able to lift a weight of 20 kg without discomfort.

Today, 5 months after the nerve block, she remains asymptomatic.

DISCUSSION

Nerve entrapment syndromes are recognized complications of abdominal surgery, in particular inguinal herniorrhaphy, appendectomy, and operations involving the Pfannenstiel incision. The nerves most commonly involved are the ilioinguinal, the iliohypogastric, and the

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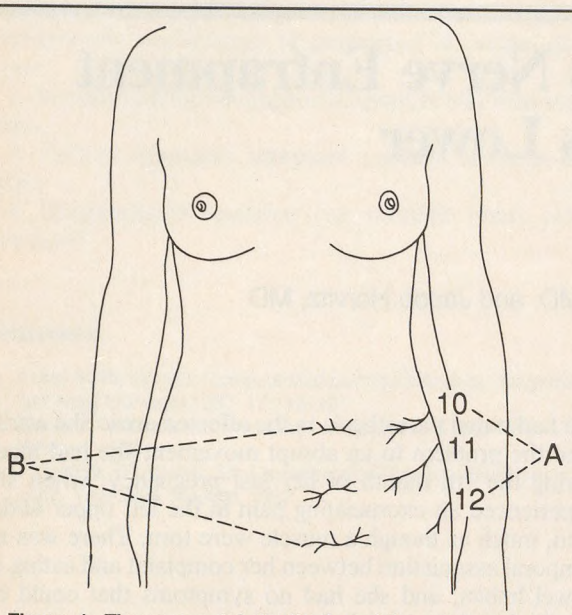

Figure 1. The segmental distribution of the cutaneous nerves: A—Lateral cutaneous branches of thoracic nerves; B—Anterior cutaneous branches of thoracic nerves.

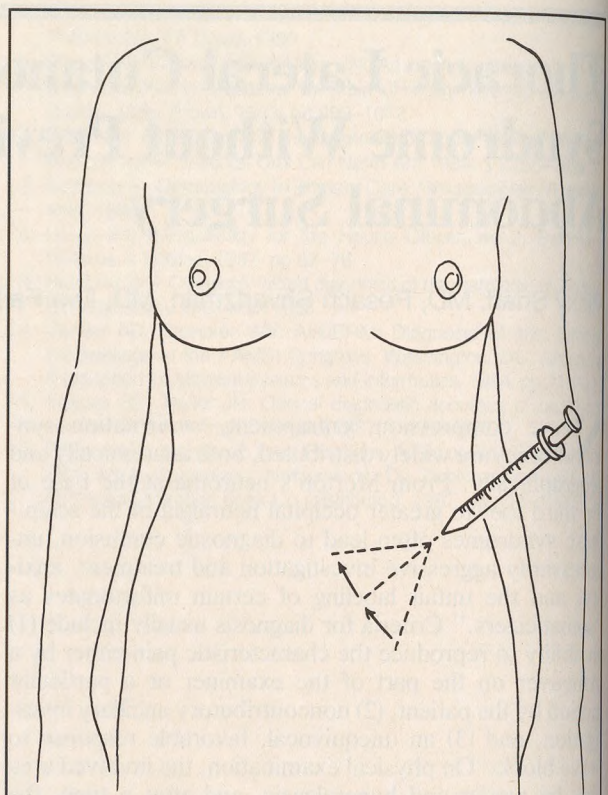

Figure 2. The pattern of injection for the nerve block is demonstrated.

genitofemoral.[4] Classically, the pain radiates from the surgical scar into the area of the affected nerve's distribution, is made worse by certain motions, and disappears at rest. With the passage of time, however, it may become fixed and take on a dull, burning character. Sufferers often receive extensive and even invasive workups aimed at the abdominal and pelvic viscera.

The pain is almost always disproportionate to the sparse physical findings, which are limited to slight tenderness on palpation and at times hypoesthesia. As a result, particularly when detailed investigations have been performed, the patient may receive an inappropriate psychiatric diagnosis. On the other hand, the dramatic response in some instances to a single peripheral nerve block, while possibly attributable to the interruption of a vicious pain-spasm pain cycle, could be interpreted as pointing to a psychogenic cause. Wright,[5] reviewing 46 patients with the slipping rib syndrome, an upper abdominal irritation neuropathy, found that most responded to reassurance alone. This modality allows the patient to come to terms with his symptoms and to feel better through knowing what he does *not* have.

Treatment for postoperative entrapment neuropathy has included, in addition to peripheral nerve block, reoperation with nerve release and paravertebral block.[4] There is no way to separate any of these from the known placebo effect of an invasive intervention. In fact, Smith,[6] in an editorial comment on a series of patients in whom excellent results were claimed for treatment of the slipping rib syndrome (a kind of nerve entrapment) by rib resection, suggested that psychiatric evaluation be part of the workup of entrapment neuropathy. He goes on to state that " . . . this diagnosis, while probably valid on occasion, must be made with due skepticism."

In the patient reported here, a therapeutic trial, whatever the mechanism of its success, carried minimal risk and seemed a small price to pay considering the 3 years of discomfort she had experienced since her injury. A referral to a psychiatrist at the time she came to the office, after many fruitless encounters with the medical profession, might have implied to her that her pain was not real and could have made her " . . . intolerant, critical and difficult."

To the author's knowledge, this instance is the first of abdominal wall nerve entrapment syndrome not associated with antecedent surgery.

The diagnosis should be kept in mind when superficial abdominal pain, aggravated by effort, has been present for an extended period and has not yielded its secret to an

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average blood glucose was 7.49 mmol/L (134.8 mg/dL; SD 1.8 mmol/L, 32.3 mg/dL), and the average cholesterol level was 5.88 mmol/L (226.3 mg/dL; SD 1.4 mmol/L, 49.5 mg/dL).

Using a two-tailed Student's *t* test, the average glycosylated hemoglobin level of these subjects was found to be higher than the 5.53% (SD 0.45%, $t = 4.45$, $P < .001$) reported for a sample of 19 normoglycemic healthy subjects with cholesterol levels less than 5.2 mmol (200 mg/dL) who took part in the early phases of this study.[6] It was also higher than 5.5% (SD 0.6%, $t = 5.44$, $P < .001$) found for 136 healthy subjects of all ages and 5.7% (SD 0.8%, $t = 3.37$, $P < .002$) found for 51 subjects between the ages of 60 and 85 years reported by the Endocrine Science group[5] using the identical methodology.

A Pearson correlation analysis was run with the variables glycosylated hemoglobin, age, total cholesterol, and glucose levels. No significant correlations were found in this group of subjects.

DISCUSSION

The results presented here demonstrate a significant elevation in glycosylated hemoglobin in nondiabetic patients suffering from a myocardial infarction compared with healthy subjects who are at low risk for atherosclerosis. The mean glycosylated hemoglobin, 6.3%, is in the low end of the range of values reported for type I (6.0% to 22%) and type II (6.2% to 20.7%) diabetics by the Endocrine Science group.[5] This elevation is unlikely to be due to the age of the patients, since Kabadi[7] found no relationship between glycosylated hemoglobin and age, and the Endocrine Science group reported only a small effect. These data are consistent with the previous report of Modan et al,[4] who found a significant elevation in HbA_{1c} in a sample of 22 patients with known myocardial infarctions. Since glycosylated hemoglobin levels measure an average of the blood glucose level for several weeks prior to the time of measurement, these results indicate that patients who suffer a myocardial infarction had glucose metabolism abnormalities that existed prior to the infarction. These results further support the evidence of Hamsten et al,[1] of Wahlberg,[2] and of Sloan et al[3] that demonstrates significant glucose metabolism abnormalities in patients with myocardial infarctions.

The modest elevation in blood glucose levels seen in these subjects reflects the stress of the acute illness. A similar acute increase in glucose levels following a myocardial infarction was reported by Ryder et al,[8] who reported an average glucose level of 7.0 mmol/L (126 mg/dL) for 58 patients within 24 hours of infarction, which returned to 5.5 mmol/L (100 mg/dL) within 9 days.

Acknowledgment

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