Microanatomy of the Structures Contributing to Abdominal Cutaneous Nerve Entrapment Syndrome

William V. Applegate, MD, and Naomi Rose Buckwalter, MD

Background: Family physicians commonly see patients who complain of abdominal pain, the cause of which cannot be readily diagnosed. Often this pain results from abdominal cutaneous nerve entrapment syndrome.

Methods: Photomicrographs were obtained of the pertinent structures of the ninth thoracolumbar nerve where it passes through the rectus muscle channel. Standard hematoxylin and eosin staining techniques were used after the paraffin autopsy tissue block was positioned to show the entire length of the rectus neurovascular bundle from anterior to posterior surfaces of the muscle (anatomically front to back). Gomori trichrome staining with aniline blue was also done to differentiate further the connective tissue structures.

Results: The photomicrographs presented here support the first author's earlier clinical and gross anatomic findings, especially regarding the fibrous ring in the rectus muscle, through which the neurovascular bundle travels. Special attention is given to the rectus fibrous ring, which is so important in producing the syndrome of abdominal cutaneous nerve entrapment. This structure is clearly delineated, and its role in the pathophysiology of this syndrome is described.

Conclusions: Three pictures presented here show the relevant microstructures involved in abdominal cutaneous nerve entrapment syndrome. The photomicrographs confirm the existence of a fibrous ring within which the neurovascular bundle can move freely. If this bundle is pushed or pulled too far from behind or in front, however, compression of the bundle against the ring causes nerve ischemia and symptoms of abdominal cutaneous nerve entrapment. Family physicians can save their patients unnecessary pain and expense by being aware of this syndrome. (J Am Board Fam Pract 1997;10:329-32.)

The first mention of abdominal wall pain simulating visceral pain was made in 1792, when Frank¹ published an article titled "Peritonitis Muscularis." In the 20th century, Janowski² and Cyriax³ both wrote about pain arising from nerves in the abdominal wall. Carnett⁴ next described in detail a syndrome he called intercostal neuralgia, the symptoms of which were caused by compromise of the abdominal cutaneous nerves. In 1972 Applegate⁵ described a syndrome that he called abdominal cutaneous nerve entrapment (ACNES), which in retrospect^{*} is remarkably similar to Carnett's description of intercostal neuralgia. On the basis of clinical and surgical observations and anatomic dissections, evidence was

presented to support the proposed pathophysiologic mechanism responsible for the clinical syndrome.⁵ A review of the syndrome is appropriate to the new information that follows.

Pathophysiology

The common pathologic insult in ACNES seems to be ischemia of the affected nerve. To understand the mechanisms of ischemia, recall that the thoracolumbar nerves, which terminate as cutaneous nerves, are anchored mainly at five locations: (1) the spinal cord, (2) where the posterior cutaneous branch originates, (3) where the lateral branch originates, (4) where the nerve makes an almost 90-degree turn to enter the rectus channel, and (5) the skin. The nerves are relatively free-floating except at these five points. In the

Submitted, revised, 20 February 1997.

From the Department of Family Practice (WVA) and the Department of Pathology (NRB), Kaiser Permanente Medical Center, San Diego. Address reprint requests to William V. Applegate, MD, 9281 Madison Ave, La Mesa, CA 91941.

This study was supported by the Southern California Permanente Medical Group Research Department, Pasadena, and the Department of Pathology, Kaiser Foundation Hospital, San Diego, CA 92120.

^{*}The 1972 article was published after an *Index Medicus* review of the years 1930 through 1970. Two years after the publication of the 1972 article, a physician from New Zealand wrote to Dr. Applegate with the information that Carnett⁴ had published an article in 1926 with remarkably similar findings. Indeed, both authors have independently described the same syndrome.



Figure 1. Diagram shows sites of cuts taken across the fibrous ring oriented anteroposteriorly in body plane: cut A, across the middle of ring; cut B, about halfway between A and C; cut C, at outer edge of ring.

rectus passage, each nerve and its vessels are surrounded by fat, binding the nerve, artery, and veins into a discrete bundle capable of functioning as a unit independent of surrounding muscle. Separating the bundle from the muscle is a fibrous ring situated about midway between the anterior surface of the rectus muscle and its posterior side, providing a smooth surface through which the bundle can slide.

From T-8 to T-12/L-1, the site of neurovascular egression from the anterior fascia of the rectus can be distinguished from surrounding muscle by palpating a somewhat oval depression running horizontally at the lateral rectus edge. A central bump is often palpated and represents the neurovascular, fatty plug. Surrounding this fatty plug at a deeper level, a smaller ring of dense tissue can be palpated and represents the fibrous ring, the demonstration of which is the subject of the current study.

The hypothesis that nerve ischemia causes ACNES symptoms is deduced from the close juxtaposition of the soft neurovascular bundle to the firm fibrous ring, which makes pressure effects from the ring seem likely at extremes of bundle motion. Indeed, digital pressure applied to an al-



Figure 2. Photomicrograph of cut A shows middle of fibrous ring, nerve exiting in fatty plug: a - artery, ad - adipocytes, ap - aponeurosis, b.p. - bridging portion of fibrous ring to edge of channel, f.p. - fatty plug, f.r. - fibrous ring, mu - muscle, n - nerve, v - vein.

ready symptomatic foramen exacerbates the pain. While observing an operation with the patient under local anesthetic, the principal author was able to elicit ACNES-like pain simply by applying mild traction to the affected nerve, which was then severed to relieve intractable pain.

Three main mechanisms of nerve ischemia have been described. Any condition that causes the abdomen to enlarge can cause the bundle to herniate through the fibrous ring. The fatty bundle bunches up against the fibrous ring from behind and compresses the vessels that supply blood to the nerve (reminiscent of what happens in trigger finger). In addition, anything that lengthens the natural course of the nerve enough stretches it taut against the unvielding fibrous ring, irritating the bundle and causing it to swell and compress its blood supply. The anterior branch is most likely to produce clinical symptoms, because nerve stretching is greatest at the point most distant from its origin (the spinal cord). Symptoms arising from the lateral and posterior branches occur less often, probably because the traction effect is less.

Finally, after the neurovascular bundle exits the rectus muscle, the nerve can be affected by scar



Figure 3. Photomicrograph of cut B shows bridging tissue between ring and edge of channel: a - artery, ad - adipocytes within channel, ap - aponeurosis, a.t. anchoring tissue for neurovascular bundle in rectus channel, b.t. - bridging tissue of fibrous ring to edge of channel, f.p. - fatty plug, f.r. - fibrous ring, mu muscle, n - nerve, p.f. - post fascia, v - vein.

tissue that might have resulted from injury or surgery. For instance, the T-12 nerve is commonly found entrapped in a suprapubic transverse herniorrhaphy, appendectomy, or hysterectomy scar, or the T-8 or T-9 nerve can be entrapped in a cholecystectomy scar. Such scars can elicit symptoms by compressing the nerve in the skin directly during scar contracture; when the unvielding scar places traction on the distal portion of the nerve, it can cause the bundle to herniate through the fibrous ring. Disparate motion between skin and underlying muscle can further herniate the nerve bundle. This process is manifested clinically when the patient states that the pain is brought on by certain motions, notably twisting or leaning forward.

Other Research

In 1986 Bjerklund-Johansen and Gran⁶ published an excellent photomicrograph of the neurovascular bundle and fatty plug viewed in the anteroposterior plane along the length of the rectus channel. The fatty plug was clearly visible and was



Figure 4. Photomicrograph of cut C shows continuity of anterior fascia (aponeurosis) with bridging tissue ring to edge of channel: a - artery, ap - aponeurosis, a.t. - anchoring tissue for neurovascular bundle in rectus channel, b.t. - bridging tissue of fibrous ring to edge of channel, f.p. - fatty plug, f.r. - fibrous ring, mu - muscle, n - nerve, p.f. - post fascia.

apparently herniating anteriorly from the rectus channel. These authors,⁶ however, questioned the existence of the fibrous ring, considered by the principal author to be so essential in producing these symptoms. To allay skepticism about this structure, we attempted to depict the fibrous ring photomicrographically. We believe the photomicrographs presented here confirm the existence of the fibrous ring in the rectus muscle.

Methods

The research was reviewed and approved by the Institutional Review Board of the Southern California Permanente Medical Group, Pasadena, California.

After we obtained permission to pursue autopsy from the next of kin, the anterior abdominal wall from the costal to the pubic level was excised from 13 consecutive adult cadavers obtained from a hospital. After routine fixation in 10 percent buffered formalin, 8 specimens were selected for further dissection of anterior abdominal cutaneous nerves T-9 to T-11, and their courses were traced from the posterodorsal (peritoneal) to the anteroventral (subcutaneous) aspect of the abdominal wall. Full-thickness, l-cm cubed block excisions were done in the transverse plane to show anterior (ventral) and posterior (dorsal) aspects of the abdominal wall and the intervening rectus channel where the neurovascular bundles traverse to their anterior exit points (Figure 1).

Results

Pictures were taken of representative tissue samples, and Figures 2, 3, and 4 show cross-sections of the fibrous ring surrounding the right ninth thoracolumbar nerve oriented as in Figure 1. Connective tissue is stained blue, and muscle is stained red.

Discussion

In the clinic an understanding of the pathophysiology of ACNES is essential for pinpointing the precipitating cause of the symptom. For example, is the mechanism push or pull? A tender spot and neuroforamen beneath an abdominal scar suggest a pull mechanism, which might be corrected by scar revision. Recent onset of abdominal bloating or weight gain suggests a push mechanism, and addressing these conditions is important for preventing further occurrence of ACNES. ACNES pain after exercise in a young and otherwise healthy person is probably caused by push (straining) and pull (twisting) and can often be treated by providing reassurance alone; providing symptomatic relief by injection with local anesthetic might be necessary, however.

ACNES is an extremely common problem and often appears semiurgently after office hours. ACNES can also affect an office patient who has already been seen by several other physicians for undiagnosed abdominal pain. The pain in this type of patient might have already been labeled functional or neurotic, and the patient is likely to display considerable (and justifiable) anxiety. When a young woman complains of pain in the ovary, ACNES arising from T-11 or T-12 should be considered a distinct possibility. Young men are more likely to ask about hernia. Gastroenterologists often receive referrals for undiagnosed abdominal pain caused by ACNES, and if they can make the diagnosis confidently, they can save the patient further unnecessary pain and expense. Of course, the presence of ACNES does not rule out serious underlying disease. For instance,

bowel cancer can cause abdominal bloating, leading to ACNES pain. If a reasonable work-up has already been done, however, and has produced negative results, and the pain is relieved by local anesthetic injection, further evaluation is probably unnecessary.

In this day of cost-effective medical care, early diagnosis of ACNES is highly cost-efficient and saves the patient lost work time, needless diagnostic procedures, and iatrogenic anxiety. If injection alone relieves the pain, repeated injection every few days is still cost-effective because each injection takes only minutes. If the pain responds to injection but recurs for several weeks, local injection of 1 to 2 mL of absolute alcohol might be curative.

Conclusions

The photomicrographs presented here confirm the existence of the fibrous ring that encircles the neurovascular bundle in the rectus muscle. The ring is situated about halfway through the muscle, originates from the aponeurosis anteriorly, and might also receive a contribution from the posterior fascia. Within this ring, the neurovascular bundle can move rather freely in the anteroposterior plane, but if pushed or pulled too far from behind or in front, compression of the bundle against the fibrous ring results in nerve ischemia and symptoms of abdominal cutaneous nerve entrapment.

Marilyn Weiss, HT, ASCP, provided technical assistance in processing the slides.

References

- 1. Frank JP. Peritonitis muscularis. 1792. Cited in: Murray GR. An address on myofibrositis as a simulator of other maladies. Lancet 1929;1:113-5.
- 2. Janowski W. Les nevralgies des parois abdominales au point de vue pratique [Neuralgia of abdominal walls]. Rev Med 1922;29:269-88.
- 3. Cyriax EF. On various conditions that may simulate the referred pains of visceral disease, and a consideration of these from the point of view of cause and effect. Practitioner 1919;102:314-22.
- 4. Carnett JB. Intercostal neuralgia as a cause of abdominal pain and tenderness. Surg Gynecol Obstet 1926;42:625-32.
- 5. Applegate WV. Abdominal cutaneous nerve entrapment syndrome. Surgery 1972;71:118-24.
- Bjerklund-Johansen TE, Gran L. Parietale abdominalsmerter [Parietal abdominal pain: abdominal cutaneous nerve entrapment syndrome]. Tidsskr Nor Lægeforen 1986;106:2151-4.