Abdominal Wall Pain: A Common Clinical Problem

Seth Sweetser, MD

From the Division of Gastroenterology and Hepatology, Mayo Clinic, Rochester, MN.

CME Activity

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Learning Objectives: On completion of this article, you should be able to (1) define abdominal wall pain, (2) outline various pathophysiologic mechanisms of abdominal wall pain, and (3) describe a focused diagnostic and treatment approach to avoid extensive and costly evaluations of abdominal wall pain.

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Abstract

Abdominal wall pain (AWP) is a common and underrecognized cause of chronic abdominal pain. The etiology of AWP varies. History and physical examination are critical to an accurate diagnosis of AWP. Trigger point injection using either a corticosteroid, a local anesthetic, or a combination of both often gives relief of pain and is of diagnostic and therapeutic value. Increased awareness of AWP as a cause of chronic, nonvisceral abdominal pain can prevent fruitless searches for intra-abdominal pathology and reduce medical costs.

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Chronic pain originating from the abdominal wall is frequently overlooked or mistaken for visceral pain, which often leads to extensive and unnecessary diagnostic testing before a diagnosis of abdominal wall pain (AWP) is established.[1,2] There are various causes of AWP, depending on which component of the abdominal wall is affected. One of the most frequent causes of AWP is the anterior cutaneous nerve entrapment syndrome (ACNES).[3] An accurate diagnosis of AWP can be made by careful elicitation of history and thorough physical examination and being

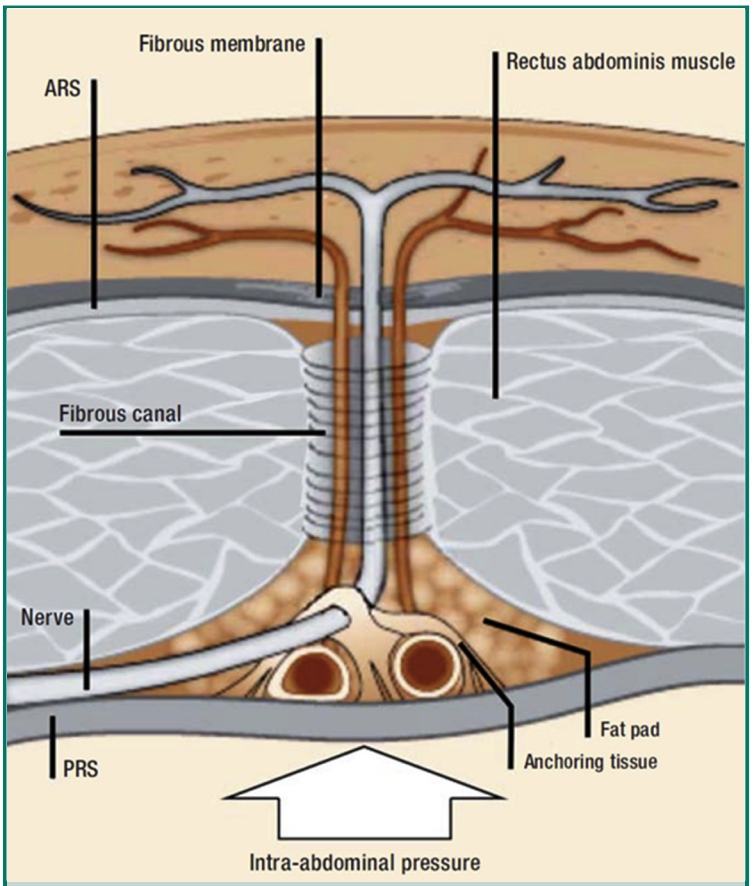

FIGURE 1. Double right-angle nerve course in anterior abdominal wall at edge of rectus abdominis muscle. ARS = anterior rectus sheath; PRS = posterior rectus sheath. From *Surg Gynecol Obstet.*[14]

alert to the possibility of symptoms arising from outside the abdominal cavity. The diagnosis of AWP can be confirmed by response to trigger point injection (TPI) of local anesthetic. Once the diagnosis is made, treatment options include conservative measures, TPI, and in refractory cases, surgery.

EPIDEMIOLOGY

Chronic AWP is common; however, estimates of its prevalence vary considerably among studies. In a primary care practice, the prevalence of chronic AWP was 3.6% for patients with a previous diagnosis of functional abdominal pain.[4] It has been reported to account for up to 30% of chronic abdominal pain cases with negative findings on prior diagnostic evaluation and for up to 10% of all gastroenterology referrals.[1] Abdominal wall pain comprised 43% of cases referred to a chronic pain clinic by gastroenterologists.[5] There are no studies evaluating the prevalence of AWP in the general population.

There are several common patient characteristics associated with chronic AWP. Patients are often obese, and women have a 4-fold greater likelihood of having AWP.[2,6] In addition to obesity, other predisposing conditions include prior abdominal surgery, pregnancy, and sports-related injuries.[7] Although AWP can occur at any age, it is more commonly reported between the ages of 30 and 50 years.[8]

PATHOPHYSIOLOGY

There are various causes of AWP, and its pathophysiology depends on which component of the abdominal wall is affected. The components of the abdominal wall include the parietal peritoneum, fat, aponeurosis, musculature, and skin, with the abdominal wall deriving its somatic sensation from anterior branches of the intercostal nerves T7 through T12. Pathologic processes affecting one or more of the abdominal wall components can lead to AWP, including herpes zoster, diabetic radiculopathy, rectus sheath hematoma, Spigelian or incisional hernias, endometriosis, cancer, and nerve entrapments.[9-12]

The most common cause of AWP is ACNES,[3] which results from entrapment of an anterior cutaneous branch of one of the thoracic nerves, T7 through T12, as it passes through the rectus abdominis muscle.[13] These nerves make 90° angles just before entering a fibrous ring through the posterior rectus sheath and immediately after passing through the anterior sheath. A discrete fat pad or plug in the neurovascular bundle allows unimpeded sliding within the fibrous ring (Figure 1).[14] Three distinct mechanisms of entrapment can occur. Enlargement of the abdomen itself can cause herniation through the fibrous ring, with subsequent trapping of the nerve resulting in ischemia and pain. In addition, enlargement of the abdomen (or anything that lengthens the course of the nerve) can also cause stretching of the nerve against the hard fibrous ring, resulting in pain. This mechanism of nerve entrapment

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is supported by AWP occurring more commonly in obese and pregnant patients.[1,7,15] The third entrapment mechanism occurs when the cutaneous nerve becomes entrapped within a scar. Entrapment of T12 is often seen after appendectomy, hysterectomy, or suprapubic transverse herniorrhaphy. Nerves T8 or T9 can become entrapped after cholecystectomy.[13] Other processes affecting the nerve, including herpes zoster, tumors, or traumatic radiculitis, might cause similar pain by different mechanisms.

CLINICAL FEATURES

Certain features suggest the pain source to be the abdominal wall (Table). The AWP may initially be sharp, followed by a dull persistent ache. It is often chronic, nagging, and nonprogressive. However, the pain may range from mild to excruciating, continuous to intermittent, with complete remissions lasting for months or years. The pain may occur anywhere in the abdomen, with the right side predominating.[1,6] Furthermore, some patients might have more than one site of pain and, rarely, may present with dull pain over a broader area. The pain is often positional and exacerbated by sitting or by lying on the affected side. Additional aggravating factors include actions that lead to tensing of the abdominal musculature, such as standing, walking, stretching, coughing, laughing, or sneezing. It is often cited that AWP can be distinguished from visceral pain on the basis of not being affected by food intake and not altered by bowel movements.[9] However, this is a misconception. Food ingestion will lead to gastric distention and an increase in intra-abdominal pressure, which will lead to reflexive abdominal wall contraction and potentially increase AWP postprandially. In addition, contraction of the abdominal muscles in the process of defecation can increase AWP associated with bowel movements, making these features unhelpful in differentiating AWP from visceral etiologies. An alternative reason for concomitant visceral symptoms in patients with AWP is the segmental relationship between affected intercostal nerves and internal organs via splanchnic chains.[16]

TABLE. Characteristic Features of Abdominal Wall Pain

1. Constant or mildly fluctuating pain
2. Located most commonly in right upper quadrant
3. Focal area of pain (≤2-cm diameter)
4. Pain increased when abdominal wall tensed (presence of Carnett sign)
5. Pain intensity often related to postural changes

A common feature of AWP is that pain may be so sharply localized and most tender over a small area of the abdominal wall (less than 2 cm in diameter), which the patient indicates by pointing and a fingertip covering the entire area. This almost always indicates that the pain originates in the abdominal wall; visceral pain cannot be so precisely localized because of the wide spinal cord overlap of the viscerosensory representation.[17] However, it is important to keep in mind that when AWP is severe, the pain may radiate diffusely.

DIAGNOSTIC APPROACH

The diagnosis of AWP is relatively straightforward by history taking and physical examination; however, the most common diagnostic pitfall is not considering the condition. A key finding on physical examination is the discrete localization of pain that allows the patient to finger point to a small area of maximum tenderness. Hershfield[18] described the "hover sign" in which the patient guards the area from light touch, sometimes by seizing the examiner's hand. The Carnett sign and associated diagnostic criteria are essential parts of the physical examination. The AWP diagnostic criteria are: (1) localized pain or a fixed location of tenderness and (2) superficial tenderness or point tenderness of 2.5 cm or less in diameter or the presence of a Carnett sign.[1]

John Carnett, a Philadelphia surgeon, recognized the diagnostic problems posed by abdominal wall lesions and developed a simple test that accurately localizes the origin of symptoms to the abdominal wall.[19] The

Carnett test is performed by palpating the abdomen of the supine patient in the usual way to elicit the area of tenderness. When the tender spot is localized, the patient is asked to contract the abdominal muscles by raising the head and trunk or lower extremities off the examining table while the examiner continues to hold pressure. Once the muscles are tensed, the patient is asked if the pain has altered. A Carnett sign consists of stable or worsening pain at the point of maximal tenderness during contraction of the abdominal wall musculature.[20] If the cause of symptoms is intra-abdominal, the tensed muscles protect the viscera and the tenderness diminishes. In contrast, if the pain originates in the abdominal wall, the tenderness increases or remains unchanged, known as the Carnett sign.

The presence of the Carnett sign strongly supports the diagnosis of AWP. Pain relief after injection of a local anesthetic (ie, TPI) is considered confirmation of the diagnosis of AWP,[1] with Sharpstone and Colin-Jones[17] concluding that a successful injection after elicitation of the Carnett sign (to diagnose AWP) is "one of the most cost effective procedures in gastroenterology." Although the Carnett test alone is 78% sensitive and 88% specific for diagnosing AWP,[1] it is important to note that there can be a high placebo effect with injections[21] and the presence of AWP does not always rule out an existing intra-abdominal source of pain. Studies have revealed an intra-abdominal source of pain in 3% to 9% patients with AWP diagnosed by the presence of a Carnett sign, with the most common cause being appendicitis.[2,22-24] In many of the cases in which a visceral source of pain was subsequently diagnosed, warning signs such as unintentional weight loss were present. Therefore, like any other sign, the presence of a Carnett sign should be interpreted in context, and the clinician should remain alert to warning symptoms or signs. Furthermore, AWP can coexist with irritable bowel syndrome and other functional gastrointestinal disorders,[25] which may complicate the clinical presentation.

The pinch test is an additional physical examination maneuver that is sensitive in detecting AWP due to ACNES. In the area of abdominal tenderness, somatosensory disturbances often exist such as hypoesthesia or hyperesthesia of the skin overlying the fixed point of maximal pain. Carnett and Bates[26] found that pinching the abdominal skin was disproportionately painful compared to the contralateral side in most patients with AWP. To perform the pinch test, the skin and subcutaneous tissue of the area with somatosensory disturbance are pinched between the index finger and the thumb. The pinch test result is considered positive if light pinching evokes a disproportionate intense pain (hyperalgesia) compared to the contralateral side. A positive pinch test result is a highly sensitive (>90%) finding of AWP due to ACNES.[27,28]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of AWP includes entities with pain of seemingly abdominal wall origin that are not successfully treated with TPI. These conditions include abdominal wall hernias, endometriosis, thoracic nerve radiculopathy, lower rib pain syndromes, and psychogenic abdominal pain.

Abdominal Wall Hermias

Abdominal wall hernias occur only at sites where the aponeurosis and fascia are not covered by striated muscle and can be subtle and difficult to detect on physical examination. Assessment requires diligent physical examination. To improve detection of abdominal wall hernias, the physical examination should be performed with the patient both standing and supine. Imaging with ultrasonography or computed tomography (CT) of the abdomen is important in diagnosis of more potentially subtle hernias such as epigastric, incisional, and Spigelian hernias.[29]

Epigastric hernias are located between the xiphoid process and umbilicus. These defects in the aponeurosis are often small and produce pain out of proportion to their size because of incarceration of preperitoneal fat.[29] Incisional hernias occur as a result of increased tension on the abdominal wall at

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sites of prior surgical incisions and can lead to AWP when only omentum becomes incarcerated in the hernia without bowel obstruction. Spigelian hernias are rare lateral, ventral, abdominal hernias that are interparietal, with the hernia sac dissecting posterior to the external oblique aponeurosis.[11] Patients often present with localized pain in the area without a bulge because the hernia lies beneath the intact external oblique aponeurosis. Spigelian hernias have a high risk of strangulation because of their smaller size and require surgical intervention. Often, CT findings are diagnostic of abdominal wall hernias; however, it is not unusual for abdominal wall defects to be overlooked on CT.[9]

Endometriosis

Endometriosis can implant between the parietal peritoneum and skin and cause AWP. Surgical procedures are the greatest risk factor for the development of abdominal wall endometriosis (AWE), with a tendency to develop within or adjacent to scars resulting from operations such as cesarean delivery or hysterectomy. AWE occurs in up to 1% of women who have undergone a cesarean delivery.[30] However, 20% of cases are spontaneous, with no surgical history.[31] The most frequent clinical presentation is that of a palpable subcutaneous mass near surgical scars associated with cyclic pain and swelling during menses. However, cyclic pain is only reported in approximately 50% of patients.[32] Therefore, AWE should be considered in all women with AWP, particularly if a mass or pain is present at a prior surgical incision. Ultrasonographic findings are often diagnostic, and surgical excision is the treatment of choice because medical therapy is not effective.[33]

Thoracic Nerve Radiculopathy

The anterior abdominal wall is innervated by the intercostal nerves (T7 through T12), so the chest and thoracic spine may be a source of abdominal pain.[17] Irritation of these intercostal nerves by spinal processes may result in abdominal pain that simulates AWP. In many patients with only minor degenerative arthritis or disk herniation, there may not be any additional symptoms to suggest spinal disease and it may be misdiagnosed as a primary abdominal wall process.[34] A helpful diagnostic clue to abdominal pain of spinal origin is posterior intercostal tenderness in the region of the vertebral transverse processes.

Lower Rib Pain Syndromes

Slipping rib syndrome is characterized by pain along the lower rib margin associated with increased mobility of the anterior costal cartilages of the 8th to 11th ribs. Greater mobility of these lower ribs can result in one rib moving over another producing pain mimicking AWP from brief entrapment of the intercostal nerve as it crosses over the costal cartilage.[17] The patient may report a snapping or clicking as the ribs move relative to one another. Diagnosis can be made by the hooking maneuver in which the clinician hooks his or her fingers under the costal margin and pulls upward.[34]

Psychogenic Abdominal Pain

Psychogenic abdominal pain can mimic AWP. Psychiatric disorders such as depression and anxiety can manifest primarily as abdominal pain. Psychogenic abdominal pain is a common cause of recurrent or persistent abdominal pain and often responds to psychiatric treatment.[35] It can be difficult to differentiate AWP from psychogenic pain with the Carnett test because results are positive in 86% of patients with psychogenic abdominal pain.[36] However, common characteristics of psychogenic pain that help in differentiation include an absence of alleviating factors and an extensive painful region with a poorly defined border. The cause of the false-positive Carnett test results in patients with psychogenic abdominal pain may be due to somatosensory amplification in which there is excessive activation and increased attention to weak stimuli.[36]

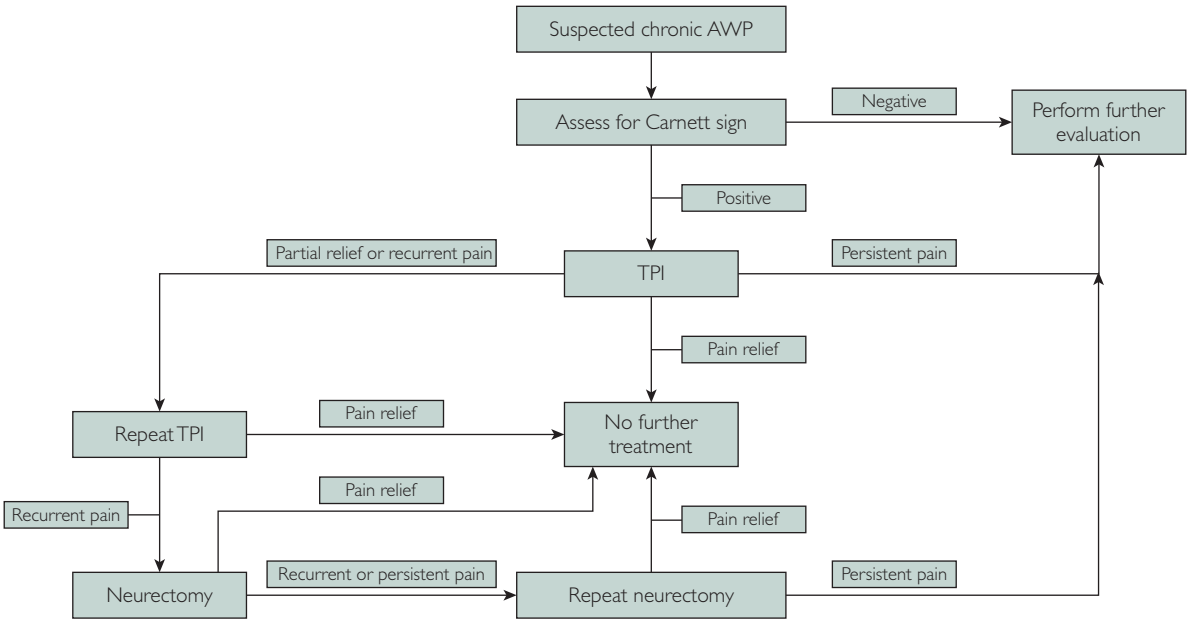

FIGURE 2. Therapeutic algorithm for chronic abdominal wall pain. TPI = trigger point injection.

TREATMENT

Multiple treatment options have been proposed for AWP. Patient education regarding the diagnosis is a treatment fundamental. An explanation of the benign etiology provides reassurance to the patient, mitigates anxiety associated with the pain, and reduces the patient's use of health care resources (eg, emergency department visits and diagnostic imaging).[2] Patients with mild-moderate AWP may choose to forgo treatment after an explanation of the diagnosis. If patients desire treatment, multiple options exist including lidocaine patch application, local injection with local anesthetic with or without corticosteroids, chemical neurolysis, and surgical neurectomy.

Various studies have focused on the outcomes of TPI with the use of corticosteroids alone or with a local anesthetic. Anesthetics are thought to interrupt a chronic pain cycle, but the reasons for long-term relief are unclear. Corticosteroids are frequently used in conjunction with the local anesthetic because they seem to enhance the anesthetic effect, perhaps by "membrane stabilization."[1] Injection of local anesthetic alone provides immediate relief in 50% to 77% of patients.[8,23,37,38] Some have reported higher success rates with combined local anesthetic and corticosteroid injections than with local anesthetic injections alone.[39] Comparisons across studies are difficult because of varying patient selection criteria and varying definitions of response to treatment. In a systematic review of the treatment of ACNES, the follow-up period of studies ranged from 12 to 72 weeks with the significant pain-relieving effect ranging from 50% to 76%.[40] An explanation for the varied response to local injection includes imprecise deposition of injection with the use of free-hand instead of ultrasound-guided injection.[37] Alnahhas et al[6] conducted a retrospective survey-based study to determine the efficacy of ultrasound-guided TPI for AWP. Ultrasound-guided TPI provided significant, long-term relief of AWP in 30% of participants, with 60% having at least some improvement. When comparing response rates with previous studies, ultrasound guidance did not seem to increase efficacy by more precise injection.[6] In examining predictors of successful response to TPI, somatization was the only negative predictor of treatment response, which reflects that a patient with

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multiple somatic symptoms is unlikely to be a good candidate for TPI regardless of the presence of the Carnett sign.[6] For patients with persistent or recurrent pain after a single injection, a second injection into the trigger point leads to lasting relief in a small percentage.[40] Overall, studies suggest that TPI with a combination of anesthetic and corticosteroid is an effective treatment for AWP, with approximately 60% of patients having some lasting pain relief.

Neurolysis by phenol injection to treat chronic AWP has been reported.[41,42] The selection of the site for injection was identified in some patients by the aid of electrical stimulation eliciting paresthesia in the painful area. Chemical neurolysis yielded only a 54% rate of permanent and total relief of pain, which is not substantially better than TPI with anesthetic and corticosteroid. A direct comparison of these 2 treatments has not been performed. Based on limited clinical experience with phenol injections, it is difficult to recommend this treatment.

Surgical treatment with neurectomy is available for medically intractable AWP. The nerve bundle at the site of maximal pain is exposed, and a small segment is excised. This procedure should be considered only in patients who have debilitating pain with only temporary relief after repeated injection treatments and when other causes of abdominal pain have been excluded. In published series of neurectomy, the percentage of patients undergoing surgical treatment varies, likely because of varying surgical indications. The primary success rate of neurectomy over the long term is reported to be high at 70%.[43] A second operation may be highly successful for those with recurrent pain.[44] Based on available data, a treatment algorithm for chronic AWP is provided (Figure 2).

CHALLENGES AND PITFALLS

Awareness of AWP is frequently lacking, and patients often undergo unnecessary and expensive diagnostic testing before a diagnosis of AWP is reached.[2,17] Therefore, clinicians worldwide require education on AWP entities to allow earlier diagnosis and treatment. Patients with AWP diagnosed at an early stage are likely to undergo fewer unnecessary procedures and may benefit from a focused treatment regimen.

The critical component of the physical examination for AWP is the Carnett test. The diagnostic accuracy of the Carnett test for identifying AWP is high,[1] but alternative causes of pain originating from the abdominal wall should not be overlooked (eg, hernia, endometriosis). Two major causes of a false-positive Carnett test result are focal peritonitis and psychogenic abdominal pain. Focal peritonitis of the anterior abdominal wall is often secondary to acute appendicitis,[2,22-24] which emphasizes the importance of not using the Carnett sign in isolation because anorexia, nausea, and fever often accompany appendicitis. When despite the presence of the Carnett sign the overall clinical picture is suggestive of a more serious underlying pathology, it is important to exclude it.

A concerted effort in medical education is needed to increase awareness that not all abdominal pain is of visceral origin. The Carnett sign is often present in patients with psychogenic abdominal pain.[35] However, the characteristics of the psychologically mediated pain typically allow differentiation from AWP. Psychogenic abdominal pain often involves an extensive painful region with no localization and no change in the severity of pain regardless of the site of palpation. Therefore, a positive Carnett test result must always be considered in the whole clinical context.

UNRESOLVED CLINICAL QUESTIONS

There are many unresolved questions regarding the prevalence, treatment, and outcomes of AWP. Future research should focus on further characterizing the epidemiology of AWP, including prevalence within the general population. This information would lead to identification of those at risk for AWP and also increase awareness of the condition. Various treatments have been used for AWP including lidocaine patches, heating pads, and neuromodulators, but no comparisons or outcomes research have

been performed on these commonly used treatment modalities. There is critical need for clinical trials comparing the efficacy and outcomes of conservative therapy, TPI, and neurectomy.

CONCLUSION

Abdominal wall pain is a common cause of chronic abdominal pain but a frequently overlooked condition. However, it can be diagnosed easily by history and physical examination. Clinicians caring for patients with abdominal pain should be aware of this condition, particularly in cases of chronic abdominal pain in which extensive diagnostic testing has not revealed an identifiable cause. First-line treatment is patient education and TPI, which is effective in most patients and can help confirm the diagnosis. Surgical neurectomy is an option for medically intractable, severe AWP.

Abbreviations and Acronyms: **ACNES** = anterior cutaneous nerve entrapment syndrome; **AWE** = abdominal wall endometriosis; **AWP** = abdominal wall pain; **CT** = computed tomography; **TPI** = trigger point injection

Potential Competing Interests: The author reports no competing interests.**The Thematic Review on Gastroenterological Diseases will continue in an upcoming issue.**

Correspondence: Address to Seth Sweetser, MD, Division of Gastroenterology and Hepatology, Mayo Clinic, 200 First St SW, Rochester, MN 55905 (sweetser.seth@mayo.edu).

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