

Hernias as a Source of Abdominal Pain: A Matter of Concern to General Surgeons, Gynecologists, and Urologists

Michael S. Kavic, MD

Editor-in-Chief

INTRODUCTION

Only a small percentage of patients with inguinal or abdominal wall hernias present with acute abdominal pain. Typically, a physical bulge is present that may or may not be uncomfortable at the time of examination. The diagnosis of hernia is usually easy, and operative management strategies are well defined. However, it would be misleading to say that discomfort rarely accompanies hernia. As a matter of fact, symptoms attributable to hernia are probably quite common. The problem is that because of the naturally overt physical presence of a hernia, health care professionals are not well versed in eliciting a history of obscure hernia symptoms. Moreover, most physicians are not skilled in searching for the physical signs that may signify an occult hernia. For some, if a hernia cannot be seen or felt, it does not exist. Nothing, of course, could be further from the truth.

BACKGROUND

Hernias are common; approximately 5% of the population will develop an abdominal wall hernia sometime during their lifetime.³

A hernia is "the protrusion of an organ, organic part, or other bodily structure through the wall that usually contains it."¹ Another important definition in the field of hernia study is that of paries or parietes. These terms (paries and parietes) refer to 'a wall' that, in an anatomic sense, can be considered to represent the wall of an organ or body cavity.² The suffix, *osis*, signifies a process, especially a disease or morbid process, that, in addition, may convey the meaning of abnormal increase in size. *Herniosis* is the process of developing a hernia.

Other terms are important in the discussion of hernias. A hernia is incarcerated when it cannot be reduced from the

hernia site. A strangulated hernia is typically an incarcerated hernia where the herniated content's blood supply has been cut off. A Richter's hernia has only a portion of the herniated bowel, usually antimesenteric surface, incarcerated within the parities of a hernia defect. A Spigelian hernia is a fully developed intraparietal hernia sited between the walls of a body cavity. Intraparietal hernias are "hidden" from external presentation by the parities (tough fascial layers) of the abdominal wall. Spigelian hernias, however, are fully developed hernias that can incarcerate and strangulate just like those hernias that present externally.

With the exception of congenital defects, which allow for spontaneous herniation, the development of a hernia probably encompasses a several-step process that involves alteration of connective tissue (abnormal collagen structure, fibroblast dysfunction, increased elastolytic enzyme levels secondary to cigarette smoking, diet, connective tissue disorders, and other factors), mechanical stress, and aging.³⁻⁵ It is important to realize that the physical presentation of a hernia protrusion is only the last step in the process of its development.

At one time, medical opinion held that hernias never occurred without the presence of a developmental diverticulum. Russell's saccular theory, in vogue during the early 20th century, rejects the view that hernia can ever be acquired in the pathological sense:

... the presence of developmental diverticulum is a necessary antecedent in every case, and we may have an open funicular peritoneum with perfectly formed muscles: we may have congenitally weak muscles with a perfectly closed funicular peritoneum, and we may have them separately or together in infinitely variable gradations.⁶

Over 80 years ago, Harrison was one of the first to refute the saccular theory.⁷ But, it remained for Read to document changes in the rectus sheath that suggested connective tissue alterations as a causal factor in hernia genesis.⁷

Perhaps the best way to conceptualize herniosis from a mechanistic perspective is to use the model for obturator hernia proposed by Gray, Skandalakis, Soria, and Rowe.⁸ In their discussion of obturator hernia, these investigators suggest that hernias evolve over several stages. The first

Department of Surgery, Northeastern Ohio Universities College of Medicine, Rootstown, Ohio, Department of Surgery, University of Pittsburgh School of medicine, Pittsburgh, Pennsylvania, USA.

Address reprint requests to: Michael S. Kavic, MD, St Elizabeth Health Center, Surgical Education, 1044 Belmont Ave, PO Box 1790, Youngstown, OH 44501-1790, USA. Telephone 330 480 3124, Fax: 330 480 3640, E-mail: Mkavic@SLS.org

© 2005 by JSLs, *Journal of the Society of Laparoendoscopic Surgeons*. Published by the Society of Laparoendoscopic Surgeons, Inc.

stage involves development of a plug or “pilot tag” of tissue from the preperitoneal connective layer. The pilot tag can be thought of as a wedge that could be “hammered” into a potential hernia space (obturator canal, inguinal ring, sciatic foramen, and others) by coughing or increased intraabdominal pressure. The second stage involves invagination of peritoneum. A nascent opening in the peritoneum develops and gradually evolves into the third stage of hernia development, an overt pocket or space of sufficient size for entrance of abdominal content. It is at this stage that most hernias become evident as a bulge or lump of the abdominal or pelvic sidewall. The hernia is visible but is usually not painful. It can get larger with coughing or the performance of valsalva-like maneuvers.

From the above, it is not hard to imagine that during the first and second stages of hernia genesis, a person could be symptomatic without the presence of intraabdominal content in a hernia defect or demonstration of an external bulge. In the first or second stage of herniosis, an obturator nerve in cases of obturator hernia, or the ilioinguinal nerve in cases of inguinal herniation, could be compressed by a pilot tag of preperitoneal connective tissue and cause symptoms of discomfort generated by nerve compression. Increased intraabdominal pressure caused by coughing, straining, or external palpation at a trigger point for that nerve would reproduce the symptoms. The pain would be neuropathic in character and present without overt external signs of herniation.

Women with obturator, sciatic, or perineal hernias can present with nonclassical symptoms of hernia. They may even present with symptoms suggestive of chronic pelvic pain.^{9,10} But, despite treatment for endometriosis, interstitial cystitis, cystitis, urethritis, adenomyosis, adhesions, and other causes of chronic pelvic pain, these patients will not be relieved of their symptoms. In these cases, the root cause is hernia, and the presence of a hernia must first be considered before it can ever be diagnosed.

SYMPTOMATOLOGY OF HERNIA –SIGNS AND SYMPTOMS

Most hernias are diagnosed by the presence of a bulge in the abdominal wall. A physical change occurs in a patient’s habitus. These persons usually have little discomfort or pain unless the hernia is incarcerated or strangulated. However, a close review of their history will reveal that many persons with hernia have experienced vague pain or discomfort particularly with physical activity.

Hernias that are reducible characteristically have few symptoms. Hernias that are incarcerated may be painful or may cause obstruction if the urinary bladder, ovary, or intestines are involved. Hernias that have strangulated are generally painful and may cause nausea, vomiting, peritoneal signs, peritonitis, sepsis, or even cardiovascular collapse.

Strangulated hernias usually present the least diagnostic dilemma and call for immediate operative intervention. The sequence of events typically results from an increased volume of content forced into the incarcerated segment of bowel. This can occur from coughing or straining, or after eating a large meal. The neck of the hernia sac, which is usually a tight fit, becomes even tighter, and venous and lymphatic congestion follows. If the hernia is not relieved, vascular engorgement and edema of the herniated content ensues. A progressive increase in the volume of the herniated content occurs along with increased pressure at the neck of the sac. Venous and arterial blood flow are arrested and ischemia results. If this process continues for a sufficient period of time, which can vary from hours to days, herniated content becomes gangrenous and necrotic. Septicemia and shock can occur. Even an inexperienced clinician will recognize that something is developing and surgical intervention is indicated.

More subtle are those hernias that spontaneously reduce, that are early in their development, or that are located in obscure sites. A paravesical hernia or an intraparietal (Spigelian) hernia that is not incarcerated can reduce spontaneously. Symptomatology secondary to obstructive phenomenon (bloating, cramping, nausea, vomiting, abdominal pain) would immediately resolve upon spontaneous reduction and the pain would “go away.” The patient would feel perfectly fine and there would be no physical signs of herniation.

CONCLUSION

It is easy to dismiss patients with atypical abdominal pain as being somatic. If it isn’t “female problems” in women, then it is “pain in their heads” when considering both sexes. But some of these patients may have intraparietal, paravesical, obturator, sciatic, perineal hernias, or first- and second-stage hernias of any location. Surgeons interested in care of the patient with abdominal and pelvic disease must be aware of the different varieties of atypical hernias and know that unusual symptoms may denote an early phase of herniosis. Otherwise, the old maxim regarding incomplete knowledge will become operative.

“What the eye doesn’t see, and the mind doesn’t know, does not exist.”¹¹

References:

1. *Webster's II New College Dictionary*. Boston, MA: Houghton Mifflin Company; 1999;519.
2. *Dorelands Illustrated Medical Dictionary*. 28th ed. Philadelphia, PA: Saunders; 1994;1234.
3. Malangoni MA, Gagliardi RJ. Hernias. In: Townsend CM, ed. *Sabiston Textbook of Surgery*. 17th ed. Philadelphia, PA: Elsevier; 2004;1199–1218.
4. Read RC. The metabolic role in the attenuation of transversalis fascia found in patients with groin herniation. *Hernia*. 2(suppl):17, 1998.
5. Bellon JM, Bujan J, Honduvilla NG, et al. Study of biochemical substrate and role of metalloproteinases in fascia transversalis from hernial processes. *Eur J Clin Invest*. 1997;27:510–516.
6. Russell RH. The saccular theory of hernia and the radical operation. *Lancet*. 1906;3:1197–1203.
7. Read RC. Attenuation of the rectus sheath in inguinal herniation. *Ann Surg*. 1974;179:610–614.
8. Gray SW, Skandalakis JE, Soria RE, Rowe JS Jr. Strangulated obturator hernia. *Surgery*. 1974;75:20.
9. Miklos JR, O'Reilly MJ, Saye WB. Sciatic hernia as a cause of chronic pelvic pain in women. *Obstet Gynecol*. 1998;91:998–1001.
10. Kavic MS. Chronic pelvic pain in females and obscure hernias. *Hernia*. 2000;4:250–254.
11. Lawrence DH. *Lady Chatterly's Lover*. Florence, Italy: Guiseppe Orioli; 1928.