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Chronic postherniorrhaphy pain following inguinal hernia surgery: Etiology, risk factors, anatomy, and treatment options

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Despite recent advances in surgical technique for inguinal hernia repair, chronic groin pain following the surgical repair of an inguinal hernia is not uncommon. Injury to the ilioinguinal, iliohypogastric, or genitofemoral nerves is the most common etiology. Specific risk factors that increase the likelihood of developing chronic groin pain have been identified. Ultrasound-guided peripheral nerve blocks may play an important role in the diagnosis and treatment of chronic groin pain. As in many pain syndromes, early diagnosis and early treatment are crucial. Evidence-based management of chronic groin pain is poorly studied, although cryotherapy and neuromodulation techniques show potential efficacy.
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Inguinal hernia repair is a common surgical procedure, with over 700,000 cases performed each year in the United States alone.[1] Chronic pain following hernia repair, or postherniorrhaphy pain (PHP), is not uncommon. PHP has been defined as "pain that is a direct consequence of a nerve lesion or a disease affecting the somatosensory system" in patients who did not have groin pain prior to an original hernia surgery or whose pain is different compared with that experienced preoperatively, present more than 6 months after surgery.[2] Although the definition is complex, this conception of PHP distinguishes chronic pain from normal postoperative pain, which typically improves as a surgical wound heals. It is frequently disabling and impairs quality of life.[3]

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Nerve damage incurred during surgery appears to be the most common cause of PHP because sensory disturbances are frequently seen in these patients.[4] Pain usually presents in the distribution of the affected nerve. The ilioinguinal nerve (IIN) is the most common neural pain generator in PHP, followed by the genitofemoral nerve (GFN), and iliohypogastric nerve (IHN). Less commonly, the lateral femoral cutaneous nerve or femoral nerve can be a source of pain after hernia surgery.[5] Nerve injury is a multifactorial event and includes mechanical or thermal injury during surgical dissection and repair, nerve entrapment from sutures, staples, mesh, and adhesions[6,7] or can be related to the inflammatory response to prosthetic mesh material.[8,9] Nerves are susceptible to injury during both open and laparoscopic repairs. In some cases, somatic pain will result from injury or damage to the pubic tubercle or deeper muscle layers, particularly when mesh is stapled or sutured in place. Recurrent hernia is another potential cause of PHP

but can usually be ruled out with radiographic studies such as magnetic resonance imaging, computed tomography, ultrasound, or herniography.[10]

Pain in the inguinal region, upper medial thigh, or genitals (penis, scrotum, or testicle in males; labium majus in females) is the common presenting complaint and is most often lancinating or burning in nature. Pain is worse with ambulation, stooping, extension of the ipsilateral hip, or twisting. Pain may be relieved with flexion of the thigh or with the recumbent position. Sexual intercourse is painful, regardless of gender, in about 5% of patients with PHP,[8,11] with a significant impact on overall functioning and quality of life.[4] In about 2% of male patients, PHP is accompanied by ejaculatory dysfunction or painful ejaculation.[12-15] Strictures in the spermatic duct from fibrous adhesions or twisting of the spermatic cord may be a cause.

Allodynia, hyperalgesia, hypesthesia, dysesthesia, or other sensory disturbances can be found on physical examination. A positive Tinel's sign or a discrete trigger point over the affected nerve is common.[16] Loss of the cremasteric reflex may be seen if the GFN nerve is injured because this nerve provides motor innervation to the cremaster muscle in males.[17] Increased concordant pain in the groin with spine extension and rotation away from the affected side may elicit signs of inguinal nerve entrapment.[8] Selective nerve blocks can be used to identify a pain-generating nerve,[18,19] although significant variation in anatomic location of these nerves has been found in cadaver studies. Therefore, blind nerve injection techniques based purely on anatomic landmarks may lead to block failure 10% to 25% of the time.[20]

Ultrasound-guided injections for PHP

Because ultrasound has become increasingly popular to improve the efficacy of peripheral nerve blocks, ultrasound-guided techniques for IIN, IHN, and GFN blockade have been detailed in the literature.[21-23] Gofeld and Christakis[21] described using a high-frequency linear transducer to scan the groin area and identify the IIN or IHN, where in the ultrasound probe is placed perpendicular to the inguinal ligament at the anterior superior iliac spine as a starting point. The oval, hypoechoic IIN and IHN are found between the internal oblique and external oblique muscle or the transverse abdominus muscle (Figure 1). The deep circumflex iliac artery seen in short axis is often in the same plane and can guide needle placement if Doppler imaging is used. Needle entry is lateral to the transducer probe with injection of 2 to 3 cc of injectate.[21]

An alternate technique has been described by Eichenberger et al[22] based on a cadaver study with nerve dissection. Probe placement is similarly perpendicular to the inguinal ligament but the nerves are scanned, visualized, and injected 5 cm cranial and posterior to the anterior superior iliac spine. In this study, the IIN and IHN were located between the internal oblique and transverse abdominus muscles in 100% of the 37 dissections performed. At this location, the 3 abdominal layers are nonaponeurotic and well defined. Additional benefits of this proximal approach are the greater size of the nerves at this location, the singular plane in which both nerves are situated,[20] and the technical ease of this technique.[21]

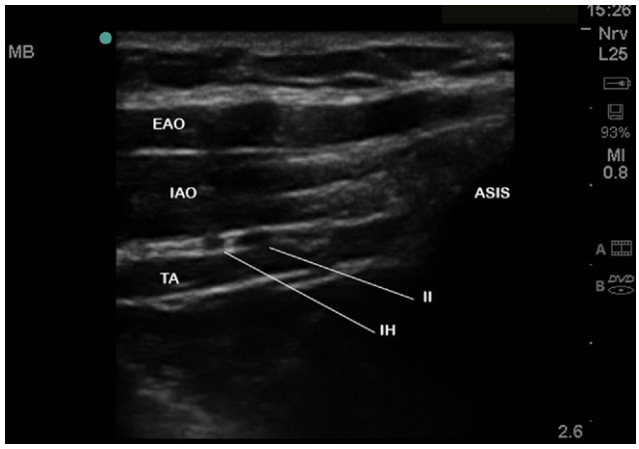

Figure 1 Ilioinguinal and iliohypogastric nerves can be identified as hypoechoic structures in the fascial plane between the internal oblique muscle and the transverse abdominus muscle. II, ilioinguinal; IH, iliohypogastric; TA, transverse abdominus muscle; IAO, internal oblique muscle; EAO, external oblique muscle; ASIS, anterior superior iliac spine. Image courtesy of Brian Chung, MD.

A prospective, randomized, double-blinded study by Weintraud et al compared ultrasound-guided IIN and IHN blocks with a blind technique in a pediatric population.[24,25] Ultrasound-guided blocks required less local anesthetic volume and evidenced dense blockade. In blindly injected subjects local anesthetic was visualized around the target nerves in only 50% compared with 100% in the treatment group.

Ultrasound-guided blockade of the GFN has been described by Peng and Tumber.[23] A high-frequency transducer is placed perpendicular to the inguinal ligament immediately lateral to the pubic tubercle, allowing visualization of the circular spermatic cord containing the GFN or the genital branch of the GFN. If the spermatic cord is not easily seen with this approach, the femoral artery can be identified as a landmark. The probe is moved in a cephalad and medial direction, bringing the spermatic cord into view (Figure 2). The use of epinephrine should be avoided with this block in men to prevent testicular ischemia. A volume of 10 cc of injectate is placed both within and around the spermatic cord. Computed tomography–guided injection of the GFN at the psoas muscle was recently described[26] and could potentially be performed with ultrasound guidance.

Increasingly, data suggest the increased success rate of peripheral nerve blockade when ultrasound guidance is used.[27] Further, lowered local anesthetic volumes are required when ultrasound guidance is used compared with

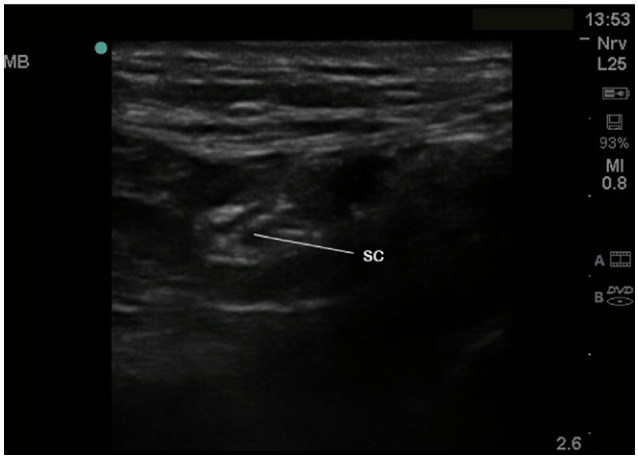

Figure 2 The genitofemoral nerve is located adjacent to or within the spermatic cord, located proximal to the inguinal crease. SC, spermatic cord. Image courtesy of Brian Chung, MD.

blind techniques using only anatomic landmarks and may translate into improved patient safety.[24]

Incidence of PHP

The literature shows wide variability in the incidence of PHP, likely because of differences in the definitions of chronic pain, onset and severity of pain, method of pain assessment, impact on functionality, and length of follow-up.

Three recent large, population-based studies with long-term follow-up show a significant incidence of this pain syndrome. Loos et al studied over 2000 patients 1 year following inguinal hernia surgery and identified moderate to severe pain in 12% of respondents with functional impairment in 20% of all cases.[28] In another large, population-based study, Bay-Nielson et al found chronic groin pain in 29% of survey respondents at 1 year following inguinal hernia surgery.[4] More than 2 years following surgery, 4% of these patients reported constant pain related to hernia surgery. Pain was described as moderate or severe in intensity in 8%. No differences in pain outcome with regard to open versus laparoscopic surgical technique, type of anesthesia, or type of hernia was identified.[4] In another study of over 5000 patients, Courtney et al found 26% of patients reporting severe pain more than 2.5 years following hernia surgery; 15% were treated in a pain clinic for PHP.[29] Massaron et al prospectively studied 1140 patients at least 10 months after hernia surgery and found 19% with chronic groin pain.[30]

In a 2007 review of the literature, a consensus panel identified an overall prevalence of PHP to be 0.5% to 6%,[2] which appears to underestimate the incidence seen in the most recent studies. There appears to be disparity between the relatively high incidence of PHP and the small population of patients treated in a pain clinic for this problem.

Anatomy

The anatomy of the 5 main nerves that supply the inguinal region is important in understanding the pathology, diagnosis, and treatment of PHP. These nerves include the IIN, IHN, GFN, lateral femoral cutaneous, and the femoral. Both the IIN and the IHN arise from the T12 and L1 anterior rami.[17] Both nerves run between the iliopsoas and quadratus lumborum muscles and pass diagonally to the abdominal wall musculature. The IHN pierces the internal oblique muscle near the anterior superior iliac spine and provides motor innervation to the internal oblique muscle as it lies between the internal and external oblique muscles. As it passes through the aponeurosis of the external oblique muscle an inch or so above the superficial inguinal ring, the IHN provides sensory fibers to the skin over the lower part of the rectus abdominis, the proximal and medial thigh, and genitals.

In a similar manner, the IIN runs parallel and below the IHN, pierces the lower border of internal oblique muscle, and then traverses to lay anterior to the spermatic cord as it traverses the inguinal region. It supplies the same areas as the IHN. In some patients, the IIN joins with the IHN or one of the nerves may be entirely absent.[31]

The L1 and L2 anterior rami form the GFN. Passing below both the IIN and the IHN from the L3 or L4 level of the psoas muscle, it eventually branches into genital or femoral branches.[31] The genital branch emerges from the transversalis fascia and then travels with the spermatic cord. It supplies sensation to the medial upper thigh and genitals (the base of the penis, upper scrotum, and testicles in men; the mons pubis and labium majus in women). It also sends motor fibers to the cremasteric muscle in men.[17] The femoral branch travels with the external iliac artery passing under the inguinal ligament to supply sensation to the anterior thigh.

The lateral femoral cutaneous nerve arises from the L2 and L3 roots and is purely sensory. It passes in a similar path as the IHN and IIN to the anterior superior iliac spine but passes under the inguinal ligament to provide sensation to the anterolateral thigh.[31]

The femoral nerve, composed of L2, L3, and L4 nerve fibers, provides primarily motor innervation to the quadriceps muscles, but sensory branches provide sensation to the groin and thigh. These small sensory branches may be susceptible to injury during inguinal surgery.[31]

There is considerable variation in the location and innervation patterns of these nerves.[15,17,32] Rab et al found interconnections among the IIN, IHN, and GFN in cadaveric dissections to be very common and only rarely were innervation patterns "normal" or classic.[33] Thus, identifying a specific nerve as a pain generator by history, physical examination, and nerve blocks with a blind-technique may yield false or misleading results.[22]

Risk factors

Risk factors for developing chronic pain following inguinal hernia surgery have been published (Table 1). Aasvang et al determined that impaired preoperative activity level, high pain

Table 1 Risk factors for PHP

Age <40
High preoperative pain scores
High postoperative pain scores
Body mass index >25
Recurrent hernia surgery
Genetic factors
Psychosocial factors

scores 1 month after surgery, and objectively measured sensory dysfunction in the groin region at 6 months after surgery increase the risk for PHP.[11] Dickinson et al found severe preoperative pain and age under 55 years to be statistically significant predictors of chronic pain.[34] Poobalan et al likewise found age <40 years as a risk factor for developing chronic pain,[18] and found that patients with a recurrent hernia experienced a 4-fold increased risk of developing chronic pain after repeat hernia surgery.[18]

Franneby et al studied 3000 patients following hernia surgery and identified young age, high level of preoperative pain, and presence of a postoperative complication (hematoma, seroma, and infection) as risk factors for chronic pain.[35] Massaron et al found young age, body mass index >25, and outpatient surgery to be significant risk factors.[30] Type of anesthesia has not been shown to be a risk factor for developing chronic pain after inguinal hernia surgery.[35]

Some have hypothesized that mesh is a risk factor for chronic pain based on experimental evidence showing myelin degeneration, edema, and fibrosis of neural tissue when in contact with propylene mesh[36]; however, long-term studies show no relationship between the use of mesh and the incidence of PHP. With limited tissue dissection and decreased potential for nerve trauma, laparoscopic hernia repair appears to have a lower incidence of chronic pain in some studies.[31] Finally, there is emerging evidence demonstrating genotype as a risk factor for developing chronic pain.[37]

Ventral hernias

Chronic abdominal wall pain following a ventral hernia repair, a much less common procedure, has a reported incidence of 23% to 28%[38-41] but has not been studied as extensively as pain following inguinal hernia repair. The etiology of pain in this scenario is typically related to the high tension of nonabsorbable sutures used in the repair[41] or from the mesh itself.[42] Patients report feelings of stiffness, restriction of movement, and decreased mobility emanating from the surgical site.[38] Risk factors for chronic pain are similar to chronic inguinal pain but also include obesity and perioperative wound infection.[41]

Treatment of PHP

Evidence-based treatment of PHP is scant in the literature. Most publications regarding treatment of PHP are case reports, case series, retrospective studies, and consensus opinions. Randomized controlled trials of any treatment for PHP are very rare in the literature. Like most pain syndromes, treatment of PHP is more successful when it precedes central sensitization.[16] Therefore, referral to a pain specialist when pain persists despite a normal postoperative recovery is an important factor in improving outcome. Pain from mild insults to peripheral nerves will likely resolve spontaneously with conservative treatment, and in mild cases of PHP, the use of the "analgesic ladder" for multimodal pharmacotherapy is appropriate.[16]

As with other chronic postoperative pain syndromes involving nerve injury or somatic pain, nonsteroidal anti-inflammatory drugs, membrane stabilizers, serotonin–norepinephrine reuptake inhibitors, *N*-methyl-D-aspartatic acid antagonists, and topical agents may be of benefit, but little has been objectively studied solely in PHP patients. Gabapentin was shown to be of benefit in limited cases of PHP[43] and may have a protective effect against chronic nerve pain when used preemptively before surgery.[44]

Efficacy of interventional treatments options for PHP has not been studied adequately, although many treatment options have been published and vary greatly. The prolonged infusion of perineural local anesthetic and clonidine for 3 weeks was successful for nearly 1 year in a patient with recalcitrant ilioinguinal neuralgia.[45] In another case series, cryoablation of the IIN or GFN resulted in decreased pain scores, decreased medication use, and increased physical activity in most patients who were treated.[46] Pulsed radiofrequency treatment of the IIN or GFN may be of benefit in PHP.[47,48] In contrast to thermal radiofrequency ablation in which nerve architecture is destroyed followed by Wallerian degeneration and likely neuroma formation, pulsed radiofrequency treatment creates an electromagnetic field around the peripheral nerve that may modulate pain transmission. Success with pulsed radiofrequency treatment of the spinal nerve roots in PHP has also been reported.[49] Other case reports describe success with peripheral nerve stimulation using subcutaneous electrodes in patient with PHP.[39] Peripheral nerve stimulation appears to be helpful for many pain syndromes, especially peripheral mononeuropathy.[50]

The surgical literature often recommends surgical exploration, removal of surgical mesh, or surgical neurectomy if conservative management of PHP fails to provide pain relief and function remains impaired after 1 year,[2,7,31] but with varying degree of success. Aasvang et al reviewed the published outcomes on surgical neurectomy and found an incidence of pain relief from 0% to 80%.[11] Most published studies on the subject of surgical neurectomy are retrospective case series with poorly defined diagnostic criteria for chronic pain in addition to other methodological problems.[9]

Conclusions

Despite recent advances in surgical technique for inguinal hernia repair, PHP remains a common sequela. Injury to the IIN, IHN, or GFN is the most common etiology. Specific risk factors that increase the likelihood of developing PHP include young age, high preoperative and postoperative pain scores, high body mass index, recurrent hernia surgery, and postoperative wound complications. Ultrasound-guided peripheral nerve blocks may play an important role in the diagnosis of PHP and aids in identifying a specific pain generator, although significant anatomic variations and innervation patterns can make this process complicated. Alternate causes of chronic groin pain, such as urological or gynecologic disease, should be ruled out. As in many pain syndromes, early diagnosis and early treatment are crucial. Cryotherapy techniques and neuromodulation, specifically peripheral nerve stimulation, may play a role in long-term management in more severe cases of PHP but require further study.

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