Review Article

Chronic neuropathic pain following inguinal hernia repair

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Abstract

Since recurrence rates have been considerably reduced with mesh repairs, chronic pain has recently become the main concern after inguinal hernia repair (IHR), with significant impact on patient satisfaction, societal cost, and quality of life. Some 31% of all patients with persistent postsurgical pain develop neuropathic pain (NP) after IHR seeing that the inguinal nerves that cross the surgical field can directly or indirectly be damaged. Because of the multiplicity of the risk factors and the complexity of the pathophysiological mechanisms, substantial attention has been devoted to the multidisciplinary approaches and to the preventive measures. More clinical trials are needed to improve the level of evidence of the use of pharmacological, surgical, and interventional procedures in both prevention and treatment of chronic NP following IHR. In this article, the current objectives are to review the incidence, risk factors, pathogenesis, diagnosis, prevention, and treatment of chronic NP after IHR.

Keywords: Chronic pain, groin, hernia repair, inguinal hernia, neuropathic pain

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INTRODUCTION

Chronic postsurgical pain is a pain that persists beyond normal healing time and lasts at least 3 months; other causes for the pain have to be excluded, in particular pain from a condition preceding the surgery.[1,2] The etiology and mechanism of chronic pain after inguinal hernia repair (IHR) is complex and includes different types of pain: neuropathic, nonneuropathic, somatic, and visceral pain.[3] Neuropathic pain (NP) after IHR develops as a consequence of a lesion affecting the somatosensory system,[4] in particular after direct damage of the inguinal nerves.[5,6] Alfieri *et al.* defined the chronic postherniorrhaphy pain as an NP arising in patients who did not have groin pain before the hernia operation, or if they did, the postoperative pain differs from the preoperative pain.[7]

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INCIDENCE

The reported incidence of chronic pain following IHR varies widely because of the nonstandardization of follow-up period, the diversity of pain-assessment methods and pain descriptors, and multiplicity of surgical procedures (open vs. laparoscopic repair and mesh vs. nonmesh repair). In a relatively recent systematic literature review, the prevalence of NP was 31% among patients with persistent postsurgical pain after IHR.[8]

RISK FACTORS

Risk factors, including patient-dependent or surgery-dependent factors, have been implicated in the development of chronic pain following IHR [Table 1]. However, in the postoperative persistent NP, particularly after IHR, predictors are not completely established, and

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Table 1: Risk factors for chronic pain after inguinal hernia repair

Preoperative factors[5,9-14]
Younger age and female gender
Preoperative pain intensity
Psychosocial vulnerability (depression, anxiety, pain expectations, catastrophizing, and hypervigilance)
Genetic predisposition (role of HLA DQB1*03:02 allele, variations in COMT and GCH1)
Repeat surgery (recurrent hernia)
Intraoperative factors[9,10,15-17]
Anesthetic technique (general anesthesia)
Nerve injury
Uses of meshes
Composition of the mesh (heavyweight meshes)
Mesh fixation (sutures, staples/tacks)
Open repair techniques
Experience of the surgeon (low volume surgical unit, duration of surgery)
Postoperative factors[12]
Acute postoperative pain severity
Early reoperation
Postoperative bleeding or infection
Radiation therapy to the operative area
Neurotoxic chemotherapy
Psychological vulnerability (depression, anxiety)

HLA: Human leukocyte antigen, HLA-DQB1 gene : Major histocompatibility complex, class II, DQ beta 1, COMT gene: Encodes the catechol-O-methyltransferase enzyme, GCH1 gene: Encodes the GTP cyclohydrolase 1 enzyme

some are only hypothesized since there is a significant lack in studies on this topic.

To identify potential predictors associated with the persistence of NP, a Delphi study was conducted among 17 experts in the field of NP. Twelve important predictors were retained; personal patient factors (depression, anxiety, pain coping, and pain catastrophizing), environmental factors (surgery as treatment for NP), functions and structure (allodynia, hyperalgesia, duration of the complaints, duration of continuing pain, and continued high pain), and participation and trait anxiety/depression as a part of health-related quality of life.[18] Otherwise, a systematic review of high-quality studies demonstrated that unlike female gender, older age was considered as a predictor for nonsurgical persistent NP.[19] Wilson *et al.* aimed to identify patient characteristics that are predictors for developing NP following breast surgery. African–American race, history of either diabetes mellitus or fibromyalgia, and treatment with chemotherapy was all associated with an increased risk of postoperative NP.[20] Whereas, in patients who underwent surgery for sarcoma of the extremities or pelvis, multiple surgeries was identified as independent predictor of postoperative NP.[21]

Nerve injury is the most important recognized risk factor in postoperative NP. In addition to the fact that the three nerves running in the inguinal canal are highly exposed to injury during groin hernia surgery, a well-documented relationship exists between nerve damage and development of a persistent NP after IHR.[9] Furthermore, open hernia repair techniques are considered as a serious factor since minimally invasive surgery contributed extremely in reducing postoperative pain incidence.[9,10,22]

Factors other than direct nerve injury are an important cause of nerve lesion and chronic NP. Although the use of meshes is considered to be the gold standard in IHR, patients continue to complain about persistent NP. In fact, contact between the mesh and nerves may induce nerve irritation because of the mesh-related inflammatory reaction and fibrosis. Furthermore, unlike within the inguinal canal, nerves in the preperitoneal space are more predisposed to the mesh-related irritation because there is no investing fascia to protect them from direct contact with the mesh.[23] After mesh implantation, the pain might be caused by the weight and composition of the material itself. Results of numerous studies on the impact of the mesh on the postsurgical pain were in favor of light meshes compared with heavy meshes.[15,16] Moreover, mesh fixation with sutures or staples has been implicated as an important cause of chronic inguinal pain (nerve entrapment). Low degree of specialization and lack of experience of the surgeon may contribute in the occurrence of postsurgical NP.[24]

The most important postoperative factor is the presence and severity of acute postoperative pain,[5,9,25] so moderate-to-severe early postoperative pain plays a major role for developing persistent pain. In 10%–50% of patients who undergo common operations, acute postoperative pain may progress to persistent pain.[5] A multi-center, prospective study conducted on 593 patients suggested that early acute postsurgical NP significantly increases the risk of persistent postsurgical NP.[26]

PATHOGENESIS

Nerve injuries

When performing IHR, the ilioinguinal nerve, the iliohypogastric nerve, and the genital branche of the genitofemoral nerve crossing the operative field can be stretched, entrapped, crushed, burned, or sectioned. The mostly encountered lesions are compression caused by sutures, staples, or mesh-related fibrosis or meshoma,[23] and transection of the predisposed nerves.

The entrapped segment of the nerve may suffer from venous stasis, intra, and perineural edema, ischemia, and eventually fibrosis and scar tissue formation.[27] In addition, peripheral nerve injuries may lead to the formation of two types of neuromas: neuroma in continuity which usually involves all degrees of nerve injury, from normal to neurotmesis, and end-bulb neuroma which occurs

anywhere a nerve is completely divided and is unopposed by another neural tissue.[28] On the other hand, when peripheral nerve tissue comes in contact with polypropylene mesh, continuous inflammatory response, axonal dilatation, myelin degeneration, endoneurial edema with thickening of both the endoneurium and perineurium, separation of myelin layers, and fibrosis result and can lead to peripheral neuropathy.[29]

Depending on the type, the site, and severity of the lesion [Table 2], the injured nerve may regenerate with possible functional recovery. Thus, neuropraxia normally full recovers in days or weeks by remyelination, while injuries of more severe grades may regenerate during months by collateral axon sprouting from the proximal axonal end to its distal target.

Mechanisms

The development of chronic NP after IHR involves different levels in the nociceptive system which comprises primary afferent pathways (Aδ, Aβ, and C fibers), second-order neurons in the dorsal horn of the spinal cord with a projection to the brain, and descending inhibitory pathways (antinociceptive system).

Peripheral sensitization

Surgical handling induces the release of molecules and inflammatory mediators such as prostaglandin, interleukins, tumor necrosis factor alpha, growth factors, neuropeptids, substance P and histamine in the injured area from nociceptors or nonneural cells (mast cells, basophils, platelets, macrophages, neutrophils, endothelial cells, keratinocytes, and fibroblasts).[30] Inflammatory actions alter the properties of peripheral terminals of high-threshold nociceptor sensory nerves by giving rise to the phosphorylation of ion channels and receptors on the nociceptor terminal membrane, reducing threshold and increasing excitability of the peripheral injured afferent nerve fibers [Figure 1a]. As a result, normally nonpainful stimuli can cause pain. This transient mechanism of acute pain is known as peripheral sensitization and disappears after regression of the inflammation and healing.[31]

Central sensitization

In some cases, when the pain persists beyond the acute phase, another important mechanism called central sensitization take place [Figure 1b]. In fact, increased nociceptive input from the periphery into the central nerve system leads to and sustains central sensitization which is a form of synaptic plasticity in the spinal cord that amplifies pain signaling. In addition, a change in the descending antinociceptive system takes part in the development of central sensitization. This kind of neuroplasticity is experienced as abnormal hyperexcitability to a normal sensory stimulus experienced in the area of injured nerves and neighboring uninjured nerves.[32,33] Three main mechanisms are implicated in central sensitization: alteration in glutamatergic neurotransmission/N-methyl-d-aspartate receptor-mediated hypersensitivity, loss of tonic inhibitory controls, and glial-neuronal interactions.[31] Furthermore, multiple studies highlighted also the role of microRNAs in molecular mechanisms of the transition from acute physiological pain to pathological persistence.[30]

Table 2: Seddon and Sunderland classification of nerve injury

Seddon	Sunderland	Injury
Neurapraxia	Grade I	Focal segmental demyelination
Axonotmesis	Grade II	Axon damaged with intact endoneurium
Axonotmesis	Grade III	Axon and endoneurium damaged with intact perineurium
Axonotmesis	Grade IV	Axon, endoneurium, and perineurium damaged with intact epineurium
Neurotmesis	Grade V	Complete nerve transection

DIAGNOSIS

Assessment of chronic neuropathic pain after inguinal hernia repair

Clinical assessment

The identification of NP is based on subjective signs and induced symptoms by tactile, thermal, and painful stimulus. Although there are no specific pain descriptors for NP, tingling, burning, and shooting are the most typical signs of pain expression.[34] Allodynia (pain due to a normally innocuous stimulus) and hyperalgesia (increased pain from a stimulus that usually provokes pain) are important objective symptoms

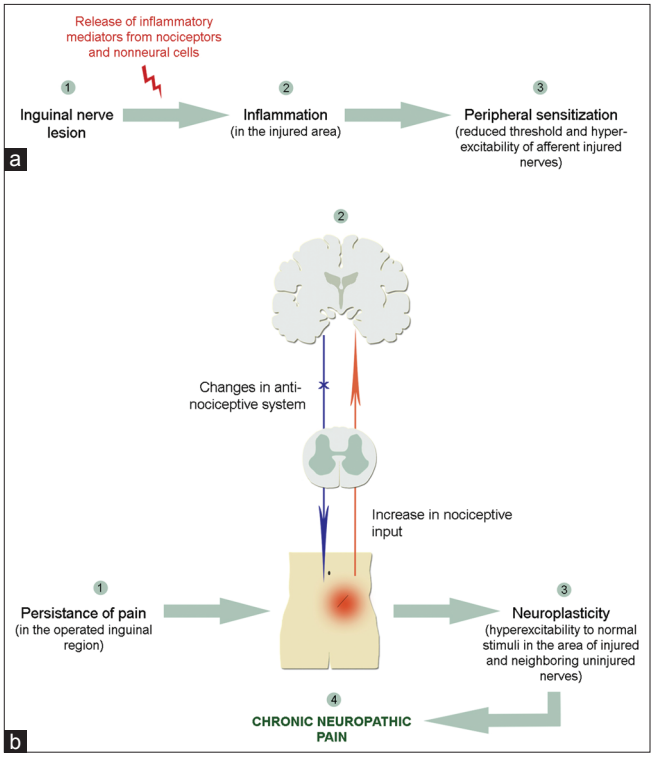

Figure 1: Mechanisms of chronic neuropathic pain. (a) Peripheral sensitization, (b) central sensitization

of NP.[32] Evenly, hypoesthesia (abnormally reduced sensation of a tactile stimulus), hypoalgesia (abnormally reduced pain sensation to a noxious stimulus), and numbness (absence of sensation) should be assessed. Frequently, both positive and negative signs are present in the same affected area; hence, the examiner may meticulously search hypoesthesia or even areas of anesthesia within painful sites which might mask the presence of a potential sensory loss area. However, sensory loss area might preclude the recognition of positive signs.[32] This phenomenon permits to distinguish NP from nociceptive pain.[33]

Physical evaluation of NP should use touch, pinprick, pressure, cold, heat, vibration, and temporal summation to map out areas of abnormal somatosensory functions. Symptoms should be sought in proximity to the inguinal scar and in the supplied territories by the three inguinal nerves (scrotum, labium, and/or upper inner thigh) keeping in mind the difficulty to identify precisely the nerve involved in causing the pain because of the anatomical varieties and connections between the nerves. In addition, clinical neurological examination of the affected area should be carefully conducted with comparison with contralateral or other unaffected body regions. Moreover, upper body stretching or twisting and/or hip joint flexing may cause pain from nerve traction or compression.[6]

Pain during sexual activity was reported in 22.1% of patients after inguinal herniorrhaphy (12.3% of genital or ejaculatory pain).[35] In a recent study, the rate of ejaculatory pain was found to be 2.2%.[36] Testicular pain that may be caused by nerve damage or testicular ischemia was encountered in 1%–6% of patients.[11] Earlier, the cremaster reflex was reported to be absent after division of the genital branch of the genitofemoral nerve.[37]

Neuropathic pain assessment tools

Numerous methods are available for screening and assessing chronic pain and discriminating between neuropathic and nonNP. For evaluating the different symptoms of NP, scales and questionnaires are developed to measure the various dimensions of pain experiences, particularly for large cohorts of patients in clinical studies [Table 3].[34] Quantitative sensory testing, a widely used tool in chronic pain assessment, uses a battery of mechanical (tactile, vibration, blunt pressure, and pinprick) and thermal (cold, heat, cold pain, and heat pain) stimuli applied to the affected area to assess disturbances in sensation that are quantified on a rating scale.[38] Thus, it allows delineating the types of nerve fibers involved, determining the threshold of detection of stimuli, diagnosing and assessing the severity of nerve damage, and helping to quantify treatment effects on NP symptoms. Laser evoked potentials have been adopted in demonstrating small-fiber involvement in numerous peripheral nervous system disorders. Recently, it is used for the investigation of the pathophysiological mechanisms of pain and the diagnosis of NP. Similarly, contact heat evoked potentials may be an objective, noninvasive diagnostic tool in small-fiber neuropathy.[39] Unfortunately, data concerning the usage of these tools in chronic NP assessment after IHR are limited. In addition, nerve conductance study is also an important tool in establishing a diagnosis of peripheral nerve lesion, but it is not easy to apply this technique on inguinal nerves because of the small caliber of the nerve fibers. Ultrasound, computed tomography scan, and MRI allow ruling out a recurrence or mesh-related factors in relation with chronic postoperative pain. Magnetic resonance neurography is effective and accurate in the clinical evaluation of peripheral nerve injury, but it is radiologist dependent and still rarely used in postherniotomy chronic pain assessment. In another technique aiming to identify which of these nerves, ilioinguinal, iliohypogastric, or genitofemoral nerve are involved, peripheral nerve block or paravertebral root block with a local anesthetic may be practiced as a diagnostic tool to determine the origin of NP.[3]

Table 3: Neuropathic pain assessment tools

The LANSS Pain Scale
The patient-based version of the LANSS Pain Scale (S-LANSS)
McGill PQ
The short-form McGill PQ
DN4
NP Questionnaire
The short form NP Questionnaire
Michigan Neuropathy Screening Instrument
NP Scale
NP Symptom Inventory
PainDETECT Questionnaire
Pain Quality Assessment Scale
ID-Pain

LANSS: Leeds assessment of neuropathic symptoms and signs, S-LANSS: Self-completed Leeds assessment of neuropathic symptoms and signs, DN4: Douleur Neuropathique en 4 questions, NP: Neuropathic pain, PQ: Pain Questionnaire

Measurement of quality of life after inguinal hernia repair

Although the patient's health-related quality of life after surgeries can be assessed using scales and questionnaires, more standardization is needed following IHR [Table 4]. Visual analog scale is the most commonly used assessment tool for pain measurement and is widely utilized in questionnaires. Persistent pain following IHR can also be assessed using a linear analog pain score (0–100; no pain = 0; mild pain <10; moderate pain 10–50; and severe pain >50).[40]

PREVENTION

Prevention of persistent NP after IHR is still a challenge because of the heterogeneity of mechanisms and the

multiplicity of risk factors. Figure 2 summarizes the key points to reduce the risk of occurrence of this complication.

Nerve handling

Since nerve trauma is the main factor of the pain, a perfect knowledge of the neuroanatomy of the groin and an increased awareness in dissection may minimize intraoperative nerve injury. During open IHR, identification and protection of the three nerves may prevent chronic NP,[10,41] while if <3 nerves are identified, more postoperative pain is present. However, a more recent prospective study found no difference in the risk of developing functional pain-related impairment in patients with and without intraoperative nerve identification.[42] To reduce the risk of nerve damage, care should be taken when incising the external oblique aponeurosis to prevent injury to the surfaced branches of the ilioinguinal or iliohypogastric nerves [Figure 3]. Usually sited in juxtaposition to the external spermatic vessels, the genital branch is protected by keeping the visible blue external spermatic vein with the spermatic cord, whereas it is being lifted from the inguinal floor.[43,44] In addition, the cremaster should be saved and incised longitudinally rather than transversely to reduce the likelihood to damage the ilioinguinal and genital branch nerve. Surgeons should avoid constricting the external ring to protect the ilioinguinal nerve from compression and avoid traumatizing the intramuscular portion of the iliohypogastric nerve by avoiding suturing the lower edge of the internal oblique muscle.[45] During laparoscopic IHRs, placement of trocar sites 2 cm above a line drawn transversely between the right and left anterior superior iliac spines (ASISs) would avoid injuring ilioinguinal and iliohypogastric nerves.[46] Surgeons should avoid deep insertion of staples or tacks and tacking of the mesh lateral to the spermatic vessels in the so-called "triangle of pain" because of the risk of injury to the femoral branch of the genitofemoral nerve or the lateral cutaneous nerve of the thigh [Figure 4]. Furthermore, to avoid nerve trauma within the inguinal canal, staples or tacks should be placed outside these at risk areas: the ilioinguinal nerve is at risk lateral to the internal ring, the genital branch of the genitofemoral nerve is at risk medial to the internal ring, and

Table 4: Questionnaires for the measurement of health-related quality of life

Short form 36 questionnaire
University of California and San Francisco Pain service patient questionnaire
Activity Assessment Scale
Pain-related sexual dysfunction after inguinal herniorrhaphy questionnaire
Inguinal PQ
Carolinas Comfort Scale
Euroqol EQ-5D

PQ: Pain Questionnaire

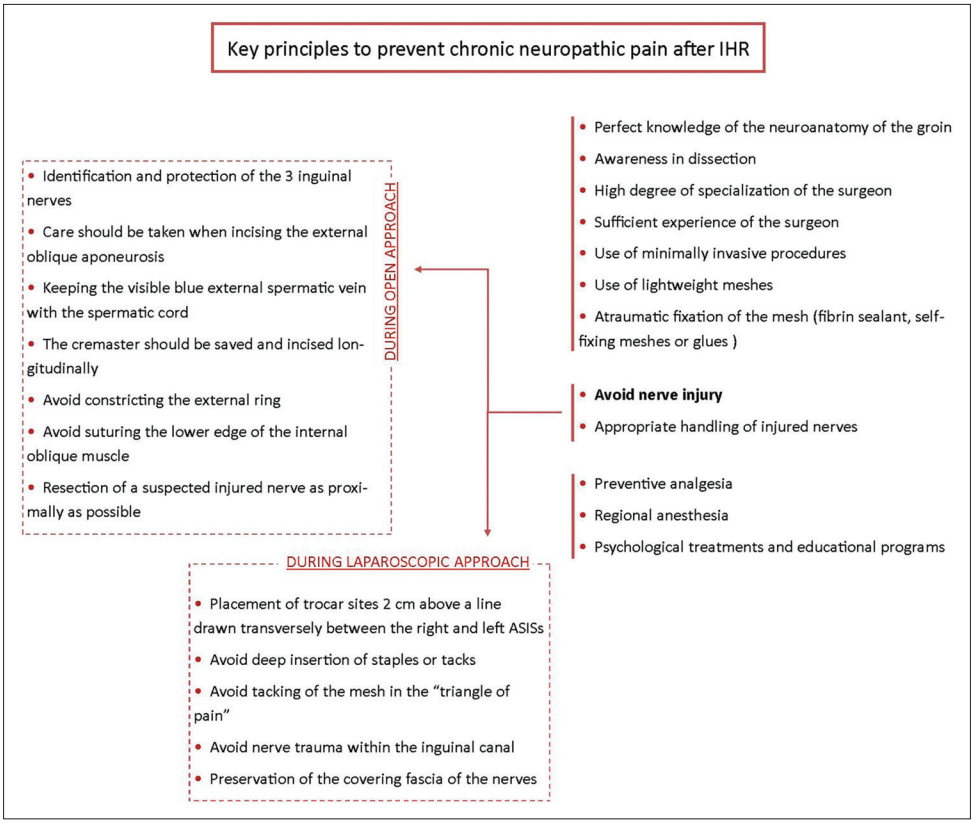

Figure 2: Prevention of chronic neuropathic pain after IHR. ASIS: anterior superior iliac spine, IHR: Inguinal hernia repair

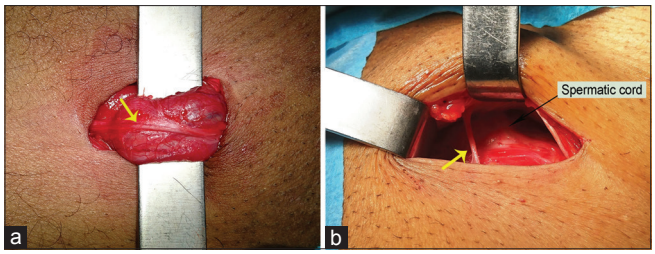

Figure 3: (a) Ilio-inguinal nerve (the spermatic cord is lifted out of the horizontal incision. Right side when performing ONSTEP procedure). (b) Ilio-hypogastric nerve (aponeurosis of the external oblique muscle is incised transversally and the caudal flap is lifted. Right side when performing ONSTEP procedure)

the iliohypogastric nerve is at risk along its entire inguinal course.[47] Evenly keeping the nerves in their natural bed and preserving, the covering fascia may reduce the risk of chronic pain.[7,45]

A suspected injured nerve or a nerve which might interfere with the positioning of the mesh should be saved, divided, or resected? In such case, it is advisable to resect the nerve as proximally as possible or under tension so that it retracts into the muscle or behind the peritoneum.[7,48] Current literature is inconsistent concerning the management of the cut ends of nerves. Hence, cut ends could be left without treatment, implanted in the muscle, crashed, ligated, cauterized, or soaked with phenol solution to prevent neuroma formation.[7]

Prophylactic neurectomy of the ilioinguinal nerve has no benefit in reducing chronic pain after open IHR and is not recommended.[22,49] However, it may lead to an increased rate of groin numbness.[50] Pragmatic division of the genital branch of the genitofemoral nerve and iliohypogastric neurectomy seems beneficial.[48,51]

Biomaterial of the mesh and mesh fixation

Meshes inducing less inflammatory reaction were designed, and light-weight meshes were compared to standard polypropylene meshes with encouraging results.[15,16] Fixation techniques of meshes are also incriminated as a cause of pain; however, there is insufficient evidence to promote fibrin sealant, self-fixing meshes, or glues ahead of suture fixation.[52,53] However, a recent meta-analysis of randomized control trials addressed in part this issue and found a lower incidence of early chronic pain in patients with biological glue fixation of the mesh in Lichtenstein IHR.[54] In laparoscopic surgery, in spite of tack or staple mesh fixation is the most widely applied technique, nonmechanical mesh fixation is considered as an alternative procedure with no real advantage in terms of chronic pain.[55,56] Otherwise, studies comparing different tack materials for mesh fixation did not show any benefit from any type of fixation.[57]

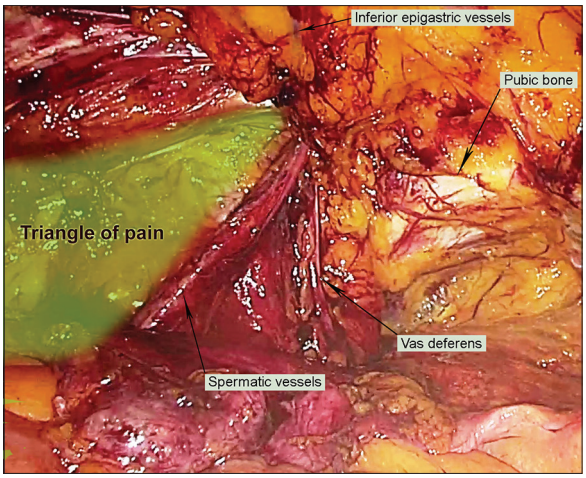

Figure 4: Triangle of pain, an area bounded superiorly by the iliopubic tract and medially by the spermatic vessels (left side when performing TAPP procedure)

Minimally invasive procedures

Minimally invasive procedures, derived from Stoppa, Rives, and Alexandre, with deep mesh implantation, are developed to lower the risk of developing chronic groin pain. Proponents of these procedures stake on the minimal dissection of the inguinal canal, the site of the mesh far from the inguinal nerves, and application of the mesh by the abdominal pressure without fixation. In the laparoscopic era, several studies have demonstrated a clear advantage of laparoscopic IHR over open repair in terms of reduced chronic postoperative pain.[58-60] However, unlike open techniques, very few researches have been interested in the type of pain after laparoscopy such as that of Bansal *et al.* in which most of the patients had non-NP character.[61] Transabdominal preperitoneal and the totally extraperitoneal approaches have similar chronic pain incidence.[58] When only considering chronic pain, endoscopic surgery is superior to open mesh for the repair of recurrent hernias.[10] In addition to the classical laparoscopic approaches, a recent study demonstrates that robotic-assisted IHR appears to be associated with low chronic pain and high health-related quality of life in the long term.[62]

Preventive analgesia

It was found in a meta-analysis that preventive analgesia was effective in reducing the incidence of chronic postsurgical NP.[63] This approach aims to block the transmission of the primary afferent injury discharge, decrease inputs into the dorsal horn and stop central sensitization. Provision of good analgesia to reduce acute postoperative pain may not completely prevent chronic pain but instead partially reduce its incidence, intensity, or duration.[64] Postoperative gabapentinoids or ketamine treatment, or infiltration

of local anesthetic might prevent the development of severe chronic pain.[3] Although there is evidence that gabapentinoids may decrease the incidence of chronic pain, there effect in hernia surgery is uncertain.[63] Sen *et al.* reported that the gabapentin-treated patients reported less intense pain at 1, 3, and 6 months after inguinal herniorrhaphy in comparison with the placebo group.[65] However, a relatively recent randomized study showed that the administration of pregabalin has no effect on preventing the development of chronic pain following inguinal hernia surgery.[66] The concept of attenuating acute postoperative pain using preemptive pre- and intra-operative analgesia in order to reduce peripheral and central sensitization need more investigation and refinement.

Regional anesthesia

In a Cochrane systematic review and meta-analysis comparing regional anesthesia versus conventional analgesia for the prevention of persistent postoperative pain, it was found that epidural anesthesia and paravertebral block, respectively, may prevent postoperative pain after thoracotomy and breast cancer surgery in about one out of every four to five patients treated.[67] The rarity of studies evaluating the effect of regional anesthesia on chronic pain following IHR does not allow the development of appropriate treatment protocols.

Psychological treatments and educational programs

A few studies related to hernia surgery that examining these issues have been published. Considering psychological risk factors, when treating pain-related fear preoperatively, the transition of acute pain to chronicity may be controlled, and the pain severity may be reduced.[68]

TREATMENT

Treatment of persistent NP after IHR is still a challenge because there is a lack of evidence-based treatment strategies. In this section, we focus on therapeutic modalities such as pharmacological, surgical, and interventional treatment that may procure sufficient pain relief [Figure 5].

Pharmacological therapy

Various guidelines recommended certain drugs for treatment of NP such as tricyclic antidepressants, selective serotonin-norepinephrine reuptake inhibitors (e.g., duloxetine and venlafaxine), calcium channel α2-δ ligands (e.g., gabapentin and pregabalin), tramadol, strong opioids although their use in chronic NP is debatable, certain antidepressant medications (e.g., citalopram and paroxetine), certain antiepileptic medications (e.g., carbamazepine, lamotrigine, topiramate, and valproic acid), and lidocaine 5% and capsaicin

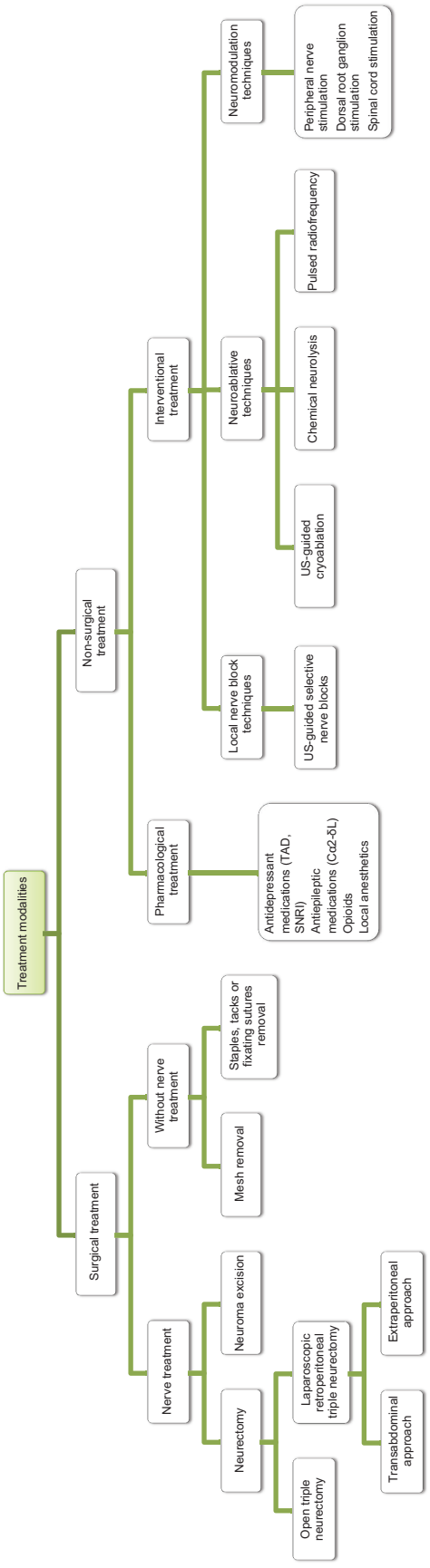

Figure 5: Therapeutic modalities of chronic neuropathic pain after IHR. TAD: Tricyclic antidepressants, SNRI: Serotonin–norepinephrine reuptake inhibitors, Cα2-δL: Calcium channel α2-δ ligands, IHR: Inguinal hernia repair

patches. So far, the utility of pharmacological treatment in the management of chronic groin postoperative pain is unclear.

Surgical therapy

Surgical treatment should be considered when NP is refractory to analgesics for at least 1 year after the initial repair, pain intensity impedes activity, and postoperative pain is different from preoperative pain.[7,69] Surgery, with both open and laparoscopic approaches, consists of resection of entrapped nerves (neurectomy), mesh removal in mesh-related pain, removal of endoscopic staples and tacks or fixating sutures, or excision of a neuroma.[10,70-72] Neurolysis and simple division of the nerves without complete resection are not recommended.[7,45]

Resection of ilioinguinal, iliohypogastric, and genital nerve was reported to be a proven surgical treatment for chronic postherniorrhaphy pain with 85% of complete resolution of pain.[23] Recently, neurectomy is the most recommended procedure and because of communications between nerves, all three nerves should be included.[7] Nerves resection is practiced through open, laparoscopic, or endoscopic approach.

Triple open neurectomy can be achieved through a two-stage operation including ilioinguinal and iliohypogastric neurectomies through an open inguinal approach and genital nerve neurectomy through a flank approach or through a one-stage procedure with resection of the three nerves within the inguinal canal.[73] As seen in preventive neurectomy, the dissected nerves should be resected as proximally and distally as possible.[45] For the iliohypogastric nerve, Amid resected the intramuscular portion of this nerve instead of cutting the nerve at the point of its emergence from the internal oblique muscle and pain relief was complete in >95%.[23] After open and laparoscopic preperitoneal mesh repair, inguinal triple neurectomy does not relieve pain due to the genitofemoral nerve irritation so an extension of the standard triple neurectomy to include the genitofemoral nerve overlying the psoas muscle was reported as a safe and effective procedure. The main trunk of the genitofemoral nerve is reached within the retroperitoneal space through an inguinal incision and a short segment of the nerve is resected.[47]

Laparoscopic retroperitoneal triple neurectomy may be performed through a transabdominal or extraperitoneal approach.[74,75] In the absence of recurrence or meshoma, laparoscopic triple neurectomy of the ilioinguinal, iliohypogastric, and genitofemoral nerve trunks in the retroperitoneal lumbar plexus was reported to be more effective than standard triple neurectomy and open extended triple neurectomy.[75] This procedure is preferred to treat pain following open and laparoscopic preperitoneal mesh repair because nerves within the preperitoneal space are not easily accessible.[7] Laparoscopy has the advantage to avoid complications of reoperation, the possibility to disrupt the prior hernia repair, and the difficulty of identifying the three inguinal nerves in the reoperative inguinal canal.[75]

Resection of the paravasal nerves within the inguinal canal seems to be a useful addition to a triple neurectomy for patients with orchialgia associated with inguinodynia.[23] However, after open or laparoscopic preperitoneal mesh repair, orchialgia has no established surgical treatment because the lamina propria of the vas deferens is not easily accessible between the mesh and the seminal vesicle.[47]

In some cases when removal of the mesh is needful, it is not necessary to remove the material completely,[45] and associate neurectomy provides more effective treatment of persistent inguinodynia.[75] Furthermore, it seems logical that treatment of NP caused by staples, tacks or fixating sutures consists of removal of these devices, particularly since laparoscopic removal of offending tacks used for mesh fixation in the preperitoneal space has been successfully practiced.[76] However, removal of meshes, entrapping sutures, or fixating devices and leaving the injured nerves may not lead to NP relief.[7]

Nonsurgical interventional therapies

There is limited evidence for the use of local nerve blocks for the treatment of persistent pain following surgery of the groin. Very few studies attempted to evaluate ultrasound-guided techniques for selective block of the ilioinguinal and iliohypogastric nerves with controversial results,[77,78] whereas for ultrasound-guided blockade of the genital branch of the genitofemoral nerve, several review articles has just described the technique. Neuroablative techniques including ultrasound-guided cryoablation which destructs selectively axons and myelin sheaths, and chemical neurolysis using phenol or alcohol have been used in the treatment of persistent postoperative pain.[79] Pulsed radiofrequency is an invasive pain treatment technique that uses electromagnetic energy applied at the vertebral level using fluoroscopically-guided localization of T12, L1, and L2 (dorsal root ganglia) or at the peripheral level (near the ASIS). Although this neuroablative technique provided efficacy in pain relief, its evidence base in the treatment of persistent pain after IHR is limited.[80,81] Peripheral nerve stimulation, dorsal root ganglion stimulation, and spinal cord stimulation are neuromodulation techniques in which small implantable electrical devices provide pain relief by producing paresthesias in the corresponding areas of pain.[82,83] Currently, this field remains poorly studied and further well-designed studies on neuromodulation for chronic pain relief after IHR should be conducted.

CONCLUSION

Although tension-free procedures are considered as the gold standard of hernia repair, persistent postoperative pain continues to be reported after mesh implantations. Management of patients with chronic NP is a common and challenging problem after IHR. The complexity of mechanisms, wide variety of causes, and heterogeneity in clinical presentation require meticulous assessment and special investigations to recognize the NP, and multidisciplinary approach to produce effective treatment. More clinical trials are needed to identify patients who are at high risk and to develop preventive treatment strategies of chronic NP following hernia surgery.

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Conflicts of interest
There are no conflicts of interest.

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