Review

An overview of the features influencing pain after inguinal hernia repair

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Abstract

Pain is a prominent issue in inguinal hernia repair research as its persisting appearance is a severe complication. The interest is also urged by the combination of a high number of repairs with an estimated risk for chronic postoperative pain of 11%. Almost every healthcare provider could encounter this complication. Pain is a complex study subject, mostly defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Various explanatory factors for pain following hernia repair have been reported. Most investigators, however, discuss only a few aspects. In the present review, these factors are collected to provide a more holistic synopsis of pain following hernia repair. It may be a resource for understanding this and other postsurgical pain.

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1. Introduction

Chronic pain after inguinal hernia repair has an estimated risk of 11% with a wide range of 0–43%. Although this estimation is the result of a systematic review, incidences varied widely between the studies included due to different definitions of pain. A widely accepted definition was provided by a Committee of the International Association for the Study of Pain: ‘Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage’. In order to consider this definition of pain in a comprehensive way different models exist. In this collective review, a straightforward sequential model was used (Fig. 1). Hernia repair encompassing dissection, use of foreign material and inflammation cause injury to tissue (nociceptive pain) and to nerves (neuropathic pain). In its transmission to the cerebral cortex, the nociceptive information can be modulated by patients’ factors and medical and cognitive pain reducing interventions. Considered as patients’ factors are genetics, age, memory, experience, mental and activity state. Anaesthesia and adjuvants represent medical pain reducing interventions. Their combined effect results in the experience of pain. This experience may become chronic with persisting stimulation or by changes in the sensory
signalling system, so-called neuroplasticity.6 This model is used to support the review of noxious stimuli of hernia repair, followed by the discussion of the medical pain reducing interventions and patients’ factors to complete the overview.

2. Nociceptor stimulation and nerve injury in inguinal hernia repair

Several components of hernia surgery bring about pain. Its effect is the combination of dissection and inflammation, causing nociceptor stimulation and nerve injury (Fig. 2). The experience of the operating team and the surgical technique could determine the extent of the dissection. Although the experience did not seem to influence the risk of postherniorrhaphy pain in at least three large studies,7–9 careful dissection to reduce the inflammatory responses is advocated.8 The minimal invasive endoscopic approach indeed is associated with less postoperative pain compared to the open approach.1,10,11 The benefits of the endoscopic approach might also be explained by less encounters with nerves. This is corroborated by the studies comparing the anterior and posterior open approach.12–15

The normal inflammation encountered in the surgical field is wound healing. Complications like infection and haematoma allow inflammatory mediators to lower the threshold of nociceptors which may enhance pain.16,17 In one randomised study, significantly less pain was described after two and six weeks in patients in whom ligation of the hernia sac was omitted compared with those who had a ligation.18 Hypothetically, the injury to the peritoneum causes a visceral component of pain through chemical stimulation of small, unmyelinated C-fibres. This hypothesis was not sustained in another report investigating peritoneal tears during endoscopic hernia repair.19 The use of a prosthetic mesh is the gold standard in hernia repair, since recurrence rates are significantly lower as in autologous repair. In experimental studies however the prosthetic material elicited a biomaterial-dependent chronic inflammatory response.20 The foreign body response to meshes resulted in axon oedema, loss of myelinated axons, peri- and endoneurial oedema and subsequent pain.21 In clinical practice, different shapes and materials are used, but no difference in pain results was observed.22 Less material or light-weighted meshes seem to result in less pain.1

Pain may also be dependent on the method of fixation. Sutures may cause ischaemia, muscle contraction or nerve damage resulting in pain. This is corroborated by the fact that removal of sutures can be an effective treatment in patients with pain.23,24 According to some authors, suturing through the periosteum of the pubic bone may elicit pain and may also result in (peri)ostitis.9 To limit the possible nociception due to fixation, some authors advocate the use of absorbable materials.8 In two trials however randomizing between non-absorbable sutures and absorbable sutures and fibrin glue no differences in pain sensation were found.25,26 In a comparable study design no significantly lower pain scores were reported when a mesh was fixated with staples instead of sutures.27

Injury to nerves or sensory transmitting systems leads to neuropathic pain. This may be caused by transection (neurectomy), by blunt or sharp dissection or as a result of entrapment. The anatomy of the nerves in the inguinal region varies widely.28 The ilioinguinal nerve is classically described as the primary cause of neuropathic pain because the nerve runs through the centre of the operative field. Also the iliohypogastric and the genitofemoral nerve are at risk in open surgery. The lateral femoral cutaneous and the femoral and obturator nerve are at risk when the operation is done laparoscopically. Routine transection of the nerves does not seem to diminish neuropathic pain,1 although a recent double-blind study found some advantages.29 Dissection of the spermatic cord can cause damage to the vas deferens and the blood supply of the testis. Damage to the somatic sacral or sympathetic nerves of the vas deferens may cause dysynergia of the ejaculatory muscles, which was reported in two of 315 patients in the study of Cunningham.9 Damage to the testicular artery causing ischaemic orchitis and injury to the veins may both result in atrophy of the testis. Injury to the genital branch of the genitofemoral nerve may also cause pain.30 Testicular pain has been reported in about 9% of all inguinal hernia repairs in a systematic review.1

3. Medical pain reducing interventions

Anaesthesia, adjuvants (cooling, warming, transcutaneous electrical nerve stimulation, acupuncture), perioperative and pre-emptive analgesia are regarded as interventions in processing nociceptive information (Fig. 3). As far as investigated
in the field of inguinal hernia repair their results are discussed below.

In a simplistic model of pain, the analgesic component of anaesthesia can be considered to block a pathway. Examples are binding to peripheral and central receptors (opiates) or inhibition of cyclo-oxygenase synthesis (NSAID). Preferably different classes of analgesics and different sites of analgesic administration are used. The concept of multimodal analgesia is to provide superior pain relief with reduced analgesic-related side effects.

Anaesthesia is used to diminish intra-operative pain. The technique can be local, locoregional or general or a combination of these techniques. In a recent review, local anaesthesia gave the most favourable results with regard to pain. Local anaesthesia was found to give a decreased systemic response as determined by c-reactive protein levels, compared to general anaesthesia. An ilioinguinal blockade additional to local infiltration as well as subfacial instead of subcutaneous injection was found to diminish pain. Better results have also been reported for the paravertebral blockade.

The influence of some non-pharmacological interventions on neuronal transmission has been studied. Cooling has been hypothesized to decrease the excitability of free nerve endings and to slow down noxious stimuli transmission whereas warming was suggested to diminish pain by lowering the local inflammatory response. Both cooling and warming were reported to diminish postoperative pain in clinical studies.

Based on the gate-control-theory, non-noxious stimuli of the perioperative field may inhibit passage of noxious stimuli, a principle that is applied in transcutaneous electrical nerve stimulation (TENS) therapy. The use of the TENS in the perioperative period of hernia repair showed however no benefit but it proved to be an effective treatment against chronic inguinal pain. Based on the same principle, analgesia can be provided with acupuncture.

As stated in the introduction, there is increasing recognition that long-term changes occur within the peripheral and central nervous system following noxious input. This neuronal plasticity may alter the response to sensory input. Roughly translated for practical use, this is the memory for pain and is the theoretical base for pre-emptive analgesia. Pre-operative and direct postoperative pain is associated with chronic pain. It is unclear whether this association is an indication of neuronal plasticity or predisposing constitution of the patient or both. The use of perioperative analgesia is anyhow to diminish direct postoperative pain, preferably established by multimodal analgesia. Treatment with analgesia before noxious stimuli cause excitation of the nociceptor is called pre-emptive analgesia. Its benefit has been shown in basic animal models. In several studies on pre-emptive analgesia in human clinical models, however, these results could not be confirmed.

4. Patients’ modulators of nociceptive information

Modulators of nociceptive information represent the patients’ characteristics or features. They are roughly divided into genetics, age, memory of pain, mental and activity state. Positioned above is the feature of experience, as it is more or less related to recognition of pain, age and mental state (Fig. 1). In this synopsis experience as a general concept comprises knowledge of pain gained through previous exposure like chronic pain syndromes or previous abdominal surgery. The given order of features is of declining consistency from invariable genetics up to changing activity state. The discussion below of each feature based on the collected literature is by no means complete.

The most obvious example of the influence of genetic disorders on pain is a gene mutation that results in dysfunction of the sodium channels in the cell resulting in insensitivity for pain. Also, there is some evidence that genetic polymorphisms are associated with a greater risk to develop chronic pain. For back pain, additive genetic effects were found to be modest contributors in male twins. Genetic variations in a gene that encodes for Catechol-O-Methyl Transferase (COMT) and the human-mu-receptor were found to be associated with differences in pain sensation. Gene therapy is already successfully applied in chronic pain treatment. Assessment of genetic profiles may be usable to predict therapy outcome.

Age is regarded as an independent inverse determinant. The elderly seem to rely predominantly on C-fibre input whereas younger adults have additional input from A-delta fibres. This results in a decreased function of the nociceptive sensation and an increased pain/heat perception threshold in the elderly. Furthermore, psychosocial variables may alter during aging and influence pain perception (experience). The lower pain scores in the elderly may be disturbed by pain measurement using the visual analogue scale, on which the elderly tend to underscore their pain.

Chronic pain is more often found after repair of recurrent hernias than after primary repair. More extensive dissections and a higher risk of nerve damage following previous hernia repair and previous lower abdominal surgery may be responsible. But pain memory may also be of importance, since more chronic pain syndromes are found in patients with severe...
inguinodynia. These patients may also suffer from pathologic pain perception. Descending pain modulation may play a key role: the pain-facilitating system may be activated but not turned off, rather promoting than inhibiting spinal neuron activity.

The mental state can be of influence on endogenous opiates and neural substrates and thereby on the sensation of pain. Catastrophizing is a disorder that leads to inability to tolerate pain or thoughts that pain is unbearable. The presence of catastrophizing thoughts might be associated with pain in hernia repair patients as it was in breast surgery and dental procedure patients. The same applies for depression, finding evidence in chronic pelvic and back pain. It remains unclear whether the pain contributes to the depression or the depression to the pain.

Anxiety is another psychological variable and can be divided into state and trait anxiety. State anxiety is a transitory state which varies in intensity and fluctuates over time, and trait anxiety can be defined as a personality disposition which remains relatively stable over time. State anxiety was found to be a predictor of postoperative pain in abdominal hysterectomy, as it was following other abdominal surgery. Anxiety and catastrophizing are reactions associated with fear of pain. Fear of pain was found to be related with the intensity of pain in a study wherein volunteers were administered a cold pressor procedure. The elevated fear of pain was hypothesized to induce avoidance behaviour, which in turn leads to a disuse syndrome, chronic disability and an exaggerated pain perception.

Interventions to the mental state, for example decreasing anxiety and subsequent pain have been investigated. Preoperative administration of anxiolytic benzodiazepines, however, had a minimal reductive effect on surgical techniques. The mutually compared degree of sensory information (emphasizes the sequence of medical procedures) yielded the strongest benefits in terms of reducing negative affect and pain reports in a meta-analysis.

Employment has been reported to be an influencing factor to chronic pain; however, it might be biased by age and physical activity.

5. Comment

The present collective review described the results of a portion of the literature on pain following hernia repair. Especially the issues on psychosocial variables, neuroimmunology, neuroanatomy and pharmacology are taken into minimal consideration in this review. These subjects are less frequently discussed in reports concerning pain following inguinal hernia repair, as the field of hernia research is focussed merely on surgical techniques. The mutually compared degree of influence on pain of the factors is difficult to determine. An impression of the value of factors mentioned in this review is shown in the flow chart.

The postoperative interval to pain assessment influences the outcome. Lower pain levels are found when follow-up is longer. Also relevant is the instrument for assessing chronic pain. In prospective hernia trials, a questionnaire is the most often used instrument. Compared to the use of a scale instrument like the visual analogue scale or verbal descriptor scale, the questionnaire reveals lower pain levels. One explanation could be a certain threshold to answer positively for pain, whereas with a scale a low score could be reported. Theoretically, the influence of the pain assessment instrument would be diminished with the use of quantitative sensory testing. In here, the sensory changes are measured objectively. Most studies found no or weak relationship with subjective pain measures. For understanding aetiology and therapeutic mechanisms however, these objective measurements could provide additional information in future studies.

Strategies for the prevention of chronic pain surpass variations in surgical techniques alone. The present review provided an insight into possible explanatory factors of pain and their mutual interference was emphasized. Regarding the different participants of the modulators, teamwork will be successful in preventing pain (Fig. 4).

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REFERENCES


