CHRONIC PAIN AFTER SURGERY: EPIDEMIOLOGY AND RISK FACTORS
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Persistent pain after surgery is both an important topic of research in its own right and at the same time a model for exploring mechanisms underlying the change from acute to chronic pain. During the last years, numerous studies have indicated a high prevalence of persistent pain after common surgical procedures. Overall, the incidence of chronic pain after surgery is estimated to lie in the range between 10% and 50%, while the estimated incidence of severe disabling pain after surgery is in the range of 2–10% (1).

Population-based data on the prevalence of persistent postsurgical pain has been scarce, but a recent study with 12,982 participants found that persistent pain in the area of surgery was reported by 40.4% of the patients (826 of 2043), moderate or severe pain by 18.3% (373 of 2043) (2). Hypoesthesia, hyperesthesia, or both was reported by 24.5% (501 of 2043). There were strong associations between sensory abnormalities and persistent pain, increasingly with higher pain intensities. One very interesting finding was the fact that of the 826 individuals reporting persistent pain in the anatomical area of surgery, only 51.0% reported chronic pain when questioned without specific reference to the surgery (2). This reveals potential large discrepancies in report of pain, depending on the questions asked and the context in which the questions are presented. Large differences between studies may indicate deficiencies in the definition of chronic (persistent) postsurgical pain. Initially chronic postsurgical pain was defined as pain that lasts at least 2 months after surgery and other causes for the pain have to be excluded, in particular pain from a condition preceding the surgery. This definition has been criticized. It is difficult to discriminate between prolongation of pain existing before surgery and new pain. Also, the time frame of 2 months has been questioned, 3-6 months would probably better reflect the natural healing after major surgery.

Several risk factors for persistent postoperative pain have been identified and can logically be divided into preoperative, intraoperative and postoperative risk factors (1,3). Better knowledge of risk factors may allow preventive strategies. The preoperative risk factors include psychosocial factors, genetic factors and preoperative pain - both pain in the area of surgery and other preoperative pain syndromes. A recent systematic review found evidence that preoperative anxiety and catastrophizing play a role in the development of CPSP (4). Knowing the bidirectional nature of relationship between pain and mood disorders, it is quite obvious that data collection must start before surgery and that the interaction with preoperative pain should be elucidated. Standardized instruments should be used as suggested in a recent review (5).

The presence of other pain syndromes before surgery seems to be a very important risk factor (6,7) In addition, many patients have inappropriate operations due to a pain syndromes like irritable bowel syndrome or back pain. Many of these patients continue to have the same pain after the operation, and in many cases their pain will be more severe.

Those patients who have severe pain and abnormal sensory changes four to six weeks after surgery are risk patients for persistent pain (8). Thus, it may be an interesting approach to treat more aggressively patients with severe acute postoperative pain and signs of neuropathic components 1-3 weeks after surgery, at a time when pain subsides in most patients.
In pain research the huge interindividual variability has been looked upon as noise, rather than a topic for study. The recent findings from twin studies, that a major part of the variability can be explained by genetic factors, has gained hope that genes increasing risk for both acute pain and for the chronification of pain can be identified. Several genes have been found to affect pain sensitivity and chronic pain in humans. Genes affecting transduction of noxious stimuli, nociceptor conduction, synaptic transmission and modulation of pain in humans have been reported, although rarely replicated. For review see LaCroix-Fralish & Mogil (9). The COMT gene, the OPRM1 gene and the CYP2D6 gene have also been linked to analgesic efficacy which may indirectly modulate persistent pain. The lack of clean phenotypes and adequately sized studies have been a limiting factor for progress.

In addition to the specific role of genetic and psychosocial factors, efforts have been made to preoperatively quantify the functional status of the nociceptive function by different nociceptive stimuli (heat, electricity, cold, etc.). The summary of these studies have clearly indicated that the pain response to preoperative stimuli may predict the acute pain response to an operation, and in a few studies also the risk for persistent pain (10). Special interest is related to the possible predictive role of the DNIC system for acute and chronic post-thoracotomy pain.

In many patients persistent postoperative pain demonstrates features of neuropathic pain and the highest incidences have been reported from procedures where major nerves trespass the surgical field like thoracotomy, breast surgery, amputation. The intraoperative nerve injury may be a significant risk factor, but additional factors may determine whether a patient with nerve injury eventually will present with present with neuropathic pain or not.

For most of the risk factors mentioned above remains to confirm whether the relationship with persistent postoperative pain is causal. Furthermore, no preventive strategy is yet fully documented, but several interventions are promising. The idea of preventive analgesia has evolved from preemptive analgesia by shifting the focus from timing of treatment to aiming at blocking noxious stimuli across the entire perioperative period. Since most interventions have potential side-effects it would be advantageous to reserve the most intense treatments to those at greatest risk only. Preoperative assessment as detailed above including tests of nociceptive function may help identify a high risk group suited for intervention.

REFERENCES


