How Does Acute Pain Become Chronic?

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Disclosure

- Nothing to Disclose

Learning Objectives

- Describe a patient-centered approach to the formulation of the patient with acute pain
- Review risk factors/predictors of chronic pain
- Identify rational treatment approaches to reduce the risk of developing chronic pain
New Chronic Pain

- Who develops it?

Case Example

- 45 y/o Korean woman s/p OTJI with foot crushed by heavy equipment for depression & disability
- Immediate reconstructive surgery for stability
- Poor compliance with physical therapy
- High levels of acute pain pre- and postop
- Treated with SAOs and acetaminophen
- Prescribed multiple agents for insomnia & anxiety
- After 6 months, referred to orthopedics for BKA
Typical Risk Factors

- Demographic variables
- Pain characteristics
- Psychological factors
- Contextual details

Demographics

- Age
- Gender
- Education
- Employment
- Health status
Pain Characteristics

- High pain intensity
- Long pain duration
- Radiation of pain
- Prior episodes of pain
- Multiple sites of pain
- Multiple somatic symptoms

Psychological Factors

- Negative emotion
- Depression
- Anxiety
- Anger
- Fear
- Stress
- Distress

- Catastrophizing
- Hypervigilance
- Self-efficacy
- Neuroticism
- Pain sensitivity
- Somatization
- ____________
Context

- Injured at work
- Work safety
- Work satisfaction
- Compensation
- Litigation
- Social support
- External attributions of responsibility

Risk Factors for New Chronic Pain

- Why does it matter?
Perspectives of New Chronic Pain

- **Diseases**
  - Pain sensitization
  - Major depression

- **Dimensions**
  - Pain modulation
    - Diffuse noxious inhibitory control (DNIC) efficiency
    - Temporal summation
  - Somatic symptoms

- **Behaviors**
  - Fear and avoidance
  - Substance use

- **Life stories**
  - Catastrophizing
  - Post-traumatic stress disorder (PTSD)

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Diseases

Syndrome → Pathology → Etiology

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Pathophysiology Pathogenesis
**Pharmacological Targets in Pain**

- **Ectopic Activity**
  - Na⁺ channel blockers
  - Ca²⁺ channel modulators
  - Glutamnergic enhancement
  - Glutaminergic inhibition

- **Central Sensitization**
  - Opioids/tramadol
  - Central α-agonists
  - NMDA antagonists
  - Anticonvulsants

- **Peripheral Sensitization**
  - NSAIDs
  - Vanilloids

- **Brain**
  - Descending Modulation
    - Central α-agonists
    - TCAs
    - SNRIs
    - Opioids/Tramadol

**Depression in Patients With Chronic Pain**

- Which one really came first?
Longitudinal Relationships

- Majority of the data support the diathesis-stress model (depression is a consequence of chronic pain)
- Treatment of depression improves pain and disability
- Directionality depends on the type of “depression”
  - Affective disorders (MDD, dysthymic disorder, bipolar)
  - Adjustment disorders, grief reactions, demoralization

Depressive disorders at baseline doubled the risk for new onset back pain 13 years later

- Severe depression (impairment) tripled the risk for incident back pain 12 years later
- Major depression + dysthymic disorder (excluding dysphoria) still increased risk for incident back pain 13 years later by 75%

**Summary of Negative Analyses**

- Current depression did not increase the risk for incident back pain; odds ratio (OR) = 1.70, (0.71, 4.08)
- Depression at baseline did not increase the risk for incident back pain 1 year later
- Back pain at baseline was not associated with depression at baseline
- Back pain at baseline was not associated with incident depression at any time point

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**Behaviors**

Drive → Choice → Learning
Fear and Avoidance

- Can we unlearn what we learn?

Patients With Substance Use Disorder

- What can we learn with a paradigm shift?

Susceptibility to Chronic Pain

- A history of substance use increases abuse of pain medications
- Cold pressor pain tolerance is ↓ in current opiate and cocaine users compared with former users
- Alcoholics and families of alcoholics have ↑ pain sensitivity and ↑ pain reduction with EtOH

**Brief Pain Inventory (BPI)**

- Patients reporting pain = 61%
- Pain intensity
  - Pain right now 5.1
  - Average 5.8
  - Worst 7.2
  - Least 4.6

**BPI Treatment**

- Receiving treatment for pain outside ATS = 14%
- Average relief provided by pain treatment = 51%
- Types of pain treatment being received:
  - Analgesics (NSAIDs, opioids): 12% (89% of treated)
  - Other (PT, blocks, epidurals): 7% (53% of treated)
- No one received adjuvant analgesics (ADs, AEDs)

NSAIDs: nonsteroidal anti-inflammatory drugs; PT, physical therapy; AEDs, antiepileptic drugs.

Dimensions

Potential → Provocation → Response

Pain Modulation

- How are we different?
Central Pain Modulation

- Endogenous analgesia system (individual trait)
- Capability assessed via the Diffuse Noxious Inhibitory Control (DNIC) test paradigm
- Lower DNIC efficiency is associated with pain
  - Healthy people with pain
  - Chronic pain syndromes
    - Primarily those postulated to be due to central sensitization
    - Fibromyalgia syndrome, multiple sclerosis, temporomandibular disorder, migraine, tension headache, irritable bowel syndrome


Incidence of Postthoracotomy Pain

- 62 patients undergoing thoracotomy
  - 38 men, mean age = 62 +/- 14 years, multiple causes
  - 36 patients → chronic pain, no med/surg predictors
- Mean follow-up = 29 +/- 17 weeks
- Acute postop pain = 49 +/- 21 (0-100 NPS)
- Chronic postop pain = 55 +/- 27 (0-100 NPS)
- Acute postop pain correlated with chronic pain
- DNIC efficiency correlated with chronic pain

NPS, numerical pain scale.
**Predictors of Postthoracotomy Pain**

- **Acute postoperative pain intensity (modifiable?)**
  - OR = 1.80 (1.28 – 2.77)
  - Change of 10 units on scale of 0 to 100

- **DNIC efficiency (dynamic preoperative trait)**
  - OR = 0.52 (0.33 – 0.77)
  - Change of 10 units on scale of -100 to +100
  - Probability of chronic post-thoracotomy pain
    - DNIC 0 → 80%; DNIC 40 → 23%; DNIC 50 → 12%
    - No correlation with acute postoperative pain (independent)


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**Somatic Symptoms**

- How do symptoms become chronic?
Somatization ↔ Chronic Pain?

- Prospective population-based follow-up survey
- 1658 people without chronic widespread pain
  - (No pain = 825; Some pain = 833)
- Somatic symptoms, psychological distress, fatigue, health anxiety, illness behavior
- 1404 respondents at 12-month follow-up
- New chronic widespread pain
  - 4.4% of men; 6.8% of women
  - One-third of new cases were men


Predictors of Chronic Pain

- 8% of people with some pain vs 2% w/o pain
- Health anxiety: NS
- Fatigue: OR = 2 (univariate only)
- Psychological distress: OR = 2 (univariate only)
- Somatic symptoms >2: OR = 4 (1.5 – 7.4)
- Illness behaviors: OR = 4 – 9 (1.8 – 22.2)
  - Frequent healthcare visits for symptoms that disrupt normal activity

Life Stories

Setting → Sequence → Outcome

Posttraumatic Stress Disorder

- What events are traumatic?
PTSD and Chronic Pain

- Criteria
  - Re-experiencing the event
  - Avoidance of reminders of the event
  - Hyperarousal

- Motor vehicle collisions → whiplash
  - Great variation across countries
  - Decreases if financial benefits are reduced
  - Rare for same magnitude collisions in other contexts
  - No dose effect of trauma intensity and probability


Pain Catastrophizing

- Why are these people so distressed?
Pain Catastrophizing (cont’d)

- An exaggerated negative mental set brought to bear during an actual or anticipated painful experience
- An expectation or worry about major negative consequences from a situation, even one of minor importance
- Multidimensional cognitive construct
  - Magnification: “I am afraid that something serious will happen.”
  - Rumination: “I cannot stop thinking about how much it hurts.”
  - Helplessness: “There is nothing I can do to reduce the intensity of the pain.”


Modifying Outcome

- Catastrophizing predicts
  - Acute pain intensity and sensitivity
  - Development of chronic pain, disability, ↓QoL

- Treatments for catastrophizing
  - Cognitive behavioral therapy and adaptive coping skills training
  - Distraction, relaxation, and imagery
  - Social support
  - Education

Conclusions

- What can really be done?

Preventing Chronic Pain

- Diseases
  - Repair and cure
- Dimensions
  - Guide and strengthen
- Behaviors
  - Extinguish and expose
- Life stories
  - Rescript and remoralize
Treatments of Predictors

- Diseases
  - Neuropathic pain and major depression
    - Antidepressants
    - Anticonvulsants
    - Augmenting agents

- Dimensions
  - Pain modulation and somatosensory amplification
    - Biofeedback and relaxation
    - Yoga, Tai Chi, Qigong
    - Cognitive-behavioral psychotherapy

Treatments of Predictors (cont’d)

- Behaviors
  - Substance use disorders and fear/avoidance
    - Group-based behavioral psychotherapy
    - Desensitization
    - Active physical therapy

- Life Stories
  - PTSD and catastrophizing
    - Support groups
    - Interpersonal psychotherapy
    - Insight-oriented psychotherapy
Case—Amputation was performed!

- Diseases
  - MDD: sertraline 300 mg/d
  - PAP: valproate 500 mg BID
- Dimensions
  - Introvert: puppy with training
  - Amputee: prosthetics + PT
- Behaviors
  - SUD: opioid taper after other txs
  - F&A: support groups (OT, amputees, church)
- Life stories
  - Marital therapy → infidelity → divorce
  - Vocational rehabilitation → RTW

Hope for Preventing Chronic Pain

- Recognizing profiles of risk for new chronic pain
- Preventing the transition from acute to chronic pain
- Treating specific causes of new chronic pain
- Addressing the nature of barriers to restoring health