Evaluation of Chronic Pain

Kenneth Nguyen, DO
Providence Physiatry
Objectives

• 1: Understand the pathophysiology of chronic pain

• 2: Clinical Evaluation of chronic low back pain/chronic pain
Simple/Nociceptive Pain

Our most basic understanding of pain

• How many patients view their pain
In reality....This is what you see clinically
Pathophysiology of Pain

- **Nociceptive Pain**
  - “Mechanical” pain
  - Sharp, well defined
  - Predictable
  - Clear aggravating and relieving factors

- **Neuropathic pain**
  - Spontaneous pain
  - Hard to describe or pain has many characteristics
    - Dull, achy
    - Burning, stinging, searing
  - Less predictable
Chronic pain = Dysregulation of CNS

- The central nervous system has a “check and balance” system, but through multiple factors, there over excitation (hyperactivity) of the pro-pain mediators.

- Peripheral sensitization is how we classically model chronic pain.

- Central pain is a more refractory form of chronic pain.
  - Injury along the spinothalamic pathway.
Peripheral vs Central Neuropathic Pain

Peripheral sensitization

Central or Post-Stroke Pain
Pathophysiology continued

- 3 order neuron system
- Periphery to Dorsal horn
- Dorsal horn to thalamus
- Thalamus to cortex
- Inhibitory pathway
Pain Fibers

- Alpha delta fibers: quick, thickly myelinated and transmit sharp pain (and temperature) that is easily discriminated.
- C fibers: unmyelinated and transmit pain.
- Alpha beta fibers: sense vibration and position.
Wind up and Central Sensitization

• Chronic pain is a learned pathway
  • Similar to “neuroplasticity” concept

• Overactivity of peripheral pain fibers causes recruitment of AMPA and NMDA receptors
  • Increases the excitation of pain carrying neurons (glutamate, Sub P, Calcitonin G related peptide (CGRP), etc...)

Impaired Ability to Suppress Pain

- Also, the descending inhibitory pathways (5HT, NOR, glycine and GABA) lose efficacy
  - Locus Ceruleus: Norepinephrine production
  - Rostral ventral medulla (RVM): GABA (decreases sensitivity of dorsal horn) and serotonin (5HT)
  - Periaqueductal Grey (PAG): 5HT
  - Glycine: Major inhibitory neurotransmitter

- “Checks and balance” is now completely dysregulated
Chronic Pain Cycle

Acute Pain:
Stimulus → Transmission → Modulation → Perception

Chronic Pain:
Stimulus → Transmission → Modulation → Perception → Peripheral Sensitization → Central Sensitization
Complex Regional Pain Model
Chronic Pancreatitis Pain Model

Central (cerebral) neuroplasticity
- Increased cerebral surface activity
- Cortical reorganization in viscero sensory areas
- Descending facilitation from the brainstem

Central (spinal) neuropathy
- Spinal sensitization
- Central hyperexcitability
- Increased referred pain area
- Temporal summation
- Generalized hyperalgesia

Peripheral extrapancreatic nociception (DRG)
- Enhanced depolarized resting potentials in DRG
- Suppression of A-type potassium current density in DRG

Peripheral intrapancreatic nociception
- Trypsin
- PAR-2, TRPV1
- Substance P, CGRP, NK1
- SP- and CGRP-fiber density
- NGF, TrkA, p75
- Artemin, GFRα3
- BDNF

Pancreatic neuropathy
- Neural damage
- Increased neural density
- Neural hypertrophy
- Pancreatic neuroplasticity
- Pancreatic neuritis
- Fractalkine-CX3CR1
- Neural remodeling
- Peripheral glial activation
Chronic Low Back Pain

• Fundamentally, non-malignant chronic pain, regardless of the etiology, is characterized by central sensitization, impaired or reduced inhibitory system, and may result in systemic problems (impaired sleep, emotional/mood changes, cognitive impairment, fatigue, etc...)
Treat the Whole Person

Diagram showing the normal adaptive response to an acute stimulation and dysfunctional responses to stimulations. The diagram includes the HPA axis, immune system, ANS, and neuroactive steroids. It highlights the importance of treating the whole person rather than just the symptoms.
Relation between Pain and Depression/Anxiety
Chronic Pain is Hard to Treat!

- Address the emotional and suffering component of pain
- R/o damage and nociceptive
  - Give the benefit of the doubt to the patient
- Team approach
- Change perception of pain
- Be open minded to treatments
- Be honest to patients
  
  Pain will be persistent if more than 2 years
Epidemiology of Low Back Pain (LBP)

- Lifetime prevalence of 84%
- Chronic symptoms in 10-15%
- 80-90% of economic resources are for the 10% who develop chronic LBP
- 1% of US adults are permanently disabled from back pain
  - Largest Predictor of disabling pain
    - Maladaptive pain coping behavior
    - Presence of non-organic signs
    - Concomitant psychiatric disease
    - Low baseline physical function
    - Low general health
Biomechanics of the Lumbar Spine

- Intervertebral disc: annulus fibrosis, not the nucleus, that absorbs shock
- Flexion loads the anterior disc, particularly flexion with rotation
- Lifting load close to body safest
- Z-joint (facet) allow for flex-ext
  - 90% occur at L4-5 and L5-S1
The Degenerative Cascade

- Tears of the annulus is the 1st sign of degenerative wear
- Loss of disc height, will put more stress on posterior elements (facet joint)
- Disc degeneration can precede facet joint disease by as much as 20 years
- Chain reaction, one level affective the levels above and below
Psychosocial Factors

• 30-40% of those with chronic low back pain have depression
• High correlation with anger and pain
  • Possibly related to deficient opioid modulation in those with high anxiety, anger, and fear reactivity

• As clinicians, try to reduce fear avoidance
  • Pain will be permanent; related to activity; exercise will damage their back
• Recognize pain catastrophizing
  • Excessively negative thoughts, high fear of movement
    • Reassure patient that they are not damaging their spine
    • Changes in beliefs account for 71% reduction in disability
HPI

• In addition to typical evaluation, r/o red flags
  • Cancer, infection, long tract signs, and fracture

• Yellow flags assc with developing chronic disabling pain
  • Catastrophic thinking, negative expectations, avoidance of normal activity, poor sleep, compensation issues, stress/anxiety, work issues, extended time off work

• Enquire about functional decline- what can’t the patient do anymore? What hobbies are affected by the pain?

• What position gives them the most comfort? What position does the patient sleep in?

• What hurts the most? Prolonged sitting, standing, or walking
Chronic pain patients often have long history!

- Past surgeries: Was it successful surgery?
- Previous treatments
  - Effective vs Not Effective
  - Previous medications that have worked or failed
- Social: Employment, Smoking, Alcohol (Hx of DUI?)
- Family: Chronic pain? What has helped family members?
Physical exam (cont)

• Tenderness, ROM, what direction causes more discomfort
• Don’t miss UMN or asymmetric reflexes
• Check for subtle signs of motor weakness ie EHL weakness for L5 radic
• Check for core/abd strength- can the person do a plank or bridge? Watch patient try to do a sit up

Is the patient able to maintain Neutral spine position or does the patient have to extend the lower back?
Nonorganic Signs

• Inappropriate tenderness that is widespread or superficial
• Pain with only simulated loading or rotation of the spine
• Inconsistent performance when testing the same thing in different positions
  • Ie FABER or SLR while sitting versus laying down
• Regional deficits in strength or sensation that do not have an anatomical basis
• Overreaction during physical exam
Diagnostic Evaluation

• Plain Xrays: Relative low sensitivity and specificity
  • I prefer to add flexion and extension views to r/o instability
    • Good for surgical screening perspective to evaluate spondylolisthesis
  • Good screening tool to r/o compression fractures

• MRI: Preferred imaging choice for disk disease
  • Can also evaluate soft tissue and musculature; Modic changes (acute=1)
    • Can determine the acuity of compression fractures
  • Adding gadolinium for post-surgical patients can help distinguish between epidural scar tissue or recurrent disc herniation
  • Overly sensitive? MRI study of patients without back pain that bulges and protrusions were common but extrusions were not.
  • Overall, must correlate between MRI and clinical findings
Further Imaging/Testing

• Computed Tomography (CT): Very useful for post-surgical patients to evaluate for osseous bridging to ensure true bony fusion

• Myelography: Usually ordered by spine surgeon as pre-surgical screening tool. Provides very clear picture of intrathecal defects caused by the spine pathology

• Bone scanning (Scintigraphy): Very sensitive to detect occult fractures, bony metastasis, and infection.

• EMG/NCS: Excellent test to look for physiological health of spinal nerves. Also important to help distinguish between entrapment neuropathies that may mask as radiculopathy

• Blood tests: ESR, CRP, CBC to r/o infection
Differential Diagnosis: Back\textgreater Leg pain

- Non-specific: 85% do not receive a specific diagnosis
  - Multi-factorial: deconditioning, poor muscle recruitment, emotional distress, arthritis, discogenic back pain
- Lumbar spondylosis: Often used for older patients with LBP
  - Facet joint point can refer to the knees or even below
  - Tight hip flexors \(\rightarrow\) increased lumbar lordosis \(\rightarrow\) posterior element stress
  - Biomechanically, lumbar extension and rotation increase facet joint forces
    - Facet loading maneuver is often documented, but diagnosis is through spinal injection
    - 15\% in younger patients, 40\% in older age groups
Low back pain > leg pain

Lumbar paraspinal muscle atrophy seen more likely in chronic low back, deconditioned patients and post-surgical patients

Facet synovitis and arthropathy
- Check for instability
Lumbar Disc Disease

• Internal Disc Disruption: External surface remains normal, but internal architecture is disrupted
  • Degradation of the nucleus pulposus and radial fissures extending to the outer third of the annulus (HIZ or high intensity zones on MRI)
  • Pain is transmitted by the sinuvertebral nerve
  • Diagnosis is through discogram and post-discography CT

• Disc Herniation: Bulge (>50% circumference) vs Herniation (<50%)
  • 95% herniations at L4-5 and L5-S1, followed by L3-4 and L2-3
    • Annulus fibrosis is weakest posterolaterally
  • Inflammatory and mechanical compression of nerve root
Disc Herniations (HNP)

- Clinical picture is variable. Some patients only have axial low back pain
- Various movements are provocative
  - Posterolateral herniation: Pain with flexion
  - Central herniation: Pain with extension
  - Lateral herniation: Pain with ipsilateral side bending
- Most do well with conservative management
  - Directional preference therapy and core stabilization for physical therapy (PT)
- Lumbar epidural is used to provide pain relief to allow patient to maximize physical therapy
Classic paracentral HNP

Far lateral HNP
Mild foraminal stenosis: some effacement of fat

Moderate foraminal stenosis: touching of the nerve root and near effacement of perineural fat
Lumbar Spinal Stenosis (LSS)

**ETIOLOGY**

- **Degenerative**: most common. Typically >60 y.o. Obesity and family hx are risk factors
- Spondylolisthesis (One vertebrae translating over the other, usually L4-5 or L5-S1)
- Mass: lipoma, synovial cyst, cancer, *epidural lipomatosis*
- Traumatic/post-operative fibrosis
- Skeletal disease: DISH (diffuse idiopathic hyperostosis)
- Congenital: dwarfism and spinal bifida
Degenerative LSS
Epidural Lipomatosis

- Another relatively common cause of LSS
  - R/o medical causes
    - Hyperlipidemia
    - Excessive steroid use?
  - Consider EMG
  - Refer to spine specialist
Clinical Presentation of LSS

- Neurogenic claudication is classic feature
  - Pain with walking/standing, relieved with rest
  - Pain 93%
  - Numbness/tingling 63%
  - Weakness 43%

- Examination
  - DTR absent in ankle 43% or knee %18
  - Weakness in 37%
  - Cauda equina uncommon
    - Absent reflexes, motor weakness, and bladder/bowel dysfunction

DIAGNOSIS:
1. Confirm with advanced imaging i.e. MRI L spine
2. I often use EMG/NCS to determine the severity
   - Look for active axonal damage
Vertebral Compression Fractures (VCF)

- 1.4 million VCF’s worldwide
  - Osteoporotic (chronic steroid users)
  - Multiple myeloma and cancer
  - Traumatic
- Screen with Xray, but confirm with MRI (STIR sequence) to examine acuity
- Brace, limit bed rest, and start Physical therapy (consider aquatic therapy)
- Secondary prevention very important to prevent further fractures
  - Must treat osteoporosis
    - Biphosphonates, and strontium ranelate (non-US)