Chronic Pain and Addiction

Launette Rieb  MSc, MD, CCFP, FCFP
Clinical Associate Professor, Dept. Family Practice, UBC
American Board of Addiction Medicine Certified

Some slides courtesy of Dr. Mark Phillips
College of Physicians & Surgeons of BC – Methadone 201
Learning Objectives

- Update understanding of pain and addiction
- Gain tips for pain Rx for methadone patients
- Minimize harm when prescribing opioids and other medications to patients with or at risk for substance dependence
- Realize when medications are not working
- Learn what else to suggest
Pain

“An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage, or both”

Complex
Subjective
Alarm
Pain is Primal

- Primitive & essential warning system
- Body has strategies to keep pain systems alert
- Feeling pain can maximize your chance at survival
- This is highly adaptive for acute pain
- Not so adaptive for chronic pain
Undertreatment of Pain

- “It is widely accepted that pain of all kinds (acute, chronic, and cancer pain) has been widely undertreated in the general population.”

- Marks & Sacher, 1973; Morgan, 1985
Undertreatment of Pain

- “Individuals with addictive disease are at special risk for suffering due to inadequate management of their pain.”

Savage, 1998
Goals of Treatment

- Reduction of pain (establish realistic expectations)
- Correction of sleep disturbance
- Reduction of anxiety and depression
- Restoration of function
- Elimination of unnecessary dependence on medications
Clinical Challenges

Drugs of Abuse and Pain Medication
Both Produce Behavioral Reinforcement
Dopamine

- The hallmark of every addictive substance is an increase in dopamine in the centres responsible for behavioral reinforcement.
- Initially one feels fulfilled/happy when using – the brain says pay attention and do it again.
- Later one becomes empty/yearning when not using.
- Ultimately it not related to “pleasure” just biologic drive.
Reward Pathway

prefrontal cortex

nucleus accumbens

VTA
First exposure to natural reward:

After repeated exposure – DA rise with cue only:

First time a better reward is received:

If expected reward isn’t received:

Cue

Reward
Addictive drugs get overvalued

First exposure to drug reward:

Next exposure to cue and drug:

After repeated exposure to cue and drug:
How Long Does the Brain Remember?

Nature Video

Cocaine Video

Amygdala not lit up

Amygdala activated
Expect overvaluation of analgesics

- Caution patients that opioids, benzos, and cannabis *trick brain systems* that evaluate expected gain
- Expect overvaluation of these medications *even in patients without true addiction*
- Plan to evaluate effectiveness of these medications by objective (functional) criteria
Clinical Challenges

What About Opioids for Pain?
Opioids

- Natural derivatives of the poppy plant are opium, morphine, codeine
- Heroin is a semi-synthetic, rest synthetics
- Bind to opioid receptors (mu, kappa, delta)
  - Relieving pain (psychological and physical)
  - Increasing DA in pleasure centres (NA & VTA)
  - Decreasing NOR in fight or flight centre (LC)
  - Affects brainstem (beware of OD with 2 opioids)
  - Can produce dysphoria
Initial binding to mu opioid receptors causes a decrease in pain transmission:
- 20-30% perceived pain reduction

However, with chronic use the body adapts to keep pain systems alert:
- Desensitizing opioid receptors (tolerance)
- Sometimes sensitizing pain pathways
- Can lead to more perceived pain
Opioids - tips

- Studies show analgesia up to 20-30%
- Yet patients and clinicians chase the fantasy of perfect analgesic control
- Opioids reduce the affective component of pain – “I still feel the pain but I don’t care”
- Suppress adrenalin release in LC, calming
- Withdrawal can be very painful (especially at sites of old injury) & drive further use
Risk of Addiction When Given Prescription Opioids

- Acute and post surgical pain studies
  - Risk < 1%
- Specialty practices for CNMP
  - Prevalence of opioid dependence 3-19%
- General population the prevalence ranges from 7-14%
- Beware: For those in recovery or already on methadone, giving opioids can trigger relapse
Risk of Addiction (or Relapse)

- Those at highest risk:
  - Active SUD
  - Past Hx of SUD
  - Family Hx of SUD
  - Active psychiatric illness
  - Past Hx of chronic pains requiring opioids

- Tight contracts, follow-up, and collateral
Random UDT Indicated for Those Treated with Opioids

- 21% of patients receiving opioids with NO aberrant behaviors had:
  - Positive urine drug screen for illicit drugs or for non-prescribed controlled substances.

- 14% of patients receiving opioids with significant aberrant behaviors had:
  - Negative urinary drug screen.

Red Flags in Patients

- Claims of lost/stolen scripts
- Constantly running out early
- Missed appts, faxed scripts
- Double doctoring
- Splitting (the only one who understands)
- Sedation/intoxication on presentation
- Using opioid to control anger, elevate mood, or calm anxiety
Red Flags in Physicians

- Often uses opioids first or second line
- No documentation of pain scales or function pre/post opioid therapy, chaotic charting
- Filling prescriptions early, faxing scripts
- Continues giving opioids despite evidence of abuse, or no evidence of increased function
- Can’t say no, need to be needed
- Active Substance Use Disorder in physician
Clinical Challenge

- How to diminish the risk of developing or worsening a substance use disorder when prescribing opioids?
Best Practice for Opioid Therapy

- Complete history, physical, differential Dx
- Risk assessment SUD, psychiatric issues
- Medication review
- Appropriate trial of non-opioid alternatives
- Treatment agreement: Visits/scripts, UDS
- Sufficient trial of opioid, establish efficacy
- Pre/post-opioid pain and function questions
- Taper off benzodiazepines first if possible
The 5As – Assessment Questions

☐ Activities of daily living
  ■ Work, self care, mobility, leisure, sport, sleep

☐ Analgesia

☐ Adverse effects

☐ Affect

☐ Aberrant drug-related behaviors

☐ + Accurate medication log

☐ + Ask relatives/friends/coworkers
In Patients at High Risk for SUD

- Prescribe only for well-defined somatic or neuropathic pain conditions
- Start with lower doses and titrate in small dose increments
- Monitor closely for signs of aberrant drug related behaviors
In Patients with Opioid Dependence

- Methadone or buprenorphine treatment
- Structured opioid therapy
- Abstinence based treatment

- Pts with any SUD - relapse triggered by:
  - Stress
  - Pain
  - Exposure to any addictive substance
Patients on Methadone or with SUD

- **Mild to moderate acute/chronic pain treatment**
  - Try non narcotic alternatives (methadone provides such a good opioid blockade)
  - High dose NSAIDs, acetaminophen, TCAs
  - Topicals, ice, exercise, stress reduction techniques

- **Severe Acute Pain Treatment**
  - In hospital get pain service or anesthesia to see
  - Neuromodulators, SNRIs, counseling, NA, AA
  - Opioid trial, time limited, function must ↑
Patients on Methadone

- For high does methadone patients remember to pick an opioid with higher receptor affinity and binding capacity
  - e.g. fentanyl patch, or iv if hospitalized
- Or one that can occupy other mu receptors
  - e.g. oxycodone oral
- Sunset clause needed
Opioids - tips

- 10% can’t metabolize codeine
- Oral meperidine ineffective and toxic met.

- Daily **short acting opioids** can increase the likelihood of addiction, opioid induced pain sensitivity, and tolerance
- Use long acting agents and little to no breakthrough when possible
When to Suggest Opioid Taper

- Patient on opioids without significant improvement in pain and function
- Safety sensitive position
- Spread of pain in the absence of disease progression - diffuse pain which can have allodynia and hyperalgesia (opioid hyperalgesia)
- Presence of substance abuse/dependence
Clinical Challenge

- What other reasons can account for a perceived need for more medication?
Mood and Magnification

Sunyata
Chronic Pain and Mood Pathways

- Serotonin (5-HT) and norepinephrine (NE) are key mediators of mood in the brain.

- 5-HT and NE are key modulatory neurotransmitters in the descending pain pathway and are part of the body’s endogenous analgesic system.
Antidepressants - TCAs

- High dose treats depression, v. sedating
- Low dose treats pain (many types of neuropathic pain and fibromyalgia) and sleep cycle disturbance - helps consolidate stage IV sleep
  - Start with nortriptyline 10 mg titrate up (150)
  - If not sedating enough use amitriptyline
  - If morning sedation take 2-3h before bed
TCAs, cont.

- Be mindful with concomitant SNRIs, SSRIs, neuromodulators, CVD, HTN
- TCAs (esp. amitriptyline) can cause: Dry mouth, postural hypotension, wt gain, sedation, urinary retention, HTN
- Less so if you start with nortriptyline, imipramine or desipramine
- Trazodone helps sleep cycle but not pain
Antidepressants - SNRIs

- In non-depressed patients:
  - **Venlafaxine** has been shown to significantly reduce pain. Beware of the withdrawal syndrome and CV effects
  - **Duloxetine** also as effective as amitriptyline at reducing pain with fewer side effects and has been approved for diabetic neuropathy and fibromyalgia. Less risk of CV effect or HTN? Risk of abuse and withdrawal

- Effects enhanced further in depressed pts
- Caution with CVD, HTN, TCAs
Antidepressants – SSRIs, DRI

- SSRIs little pain relieving benefit, accept perhaps escitalapram for neuropathic pain
- Primarily these agents should be used when a major mood disorder is adding to the pain picture and SNRIs have been tried or are contraindicated
- Paroxetine and citalopram are other options
- The DNRI bupropion can also assist mood
Neuromodulators (anticonvulsants)

- Initial reports of efficacy of gabapentin in post herpetic neuralgia and diabetic neuropathy 23% of pts improve, NNT=4, Cochrane Review
- Therapeutics Initiative reviewed unpublished & published studies for gabapentin and pregabalin
- **Gabapentin** induced pain reduction was more modest (<1/10), helps 15% of pts above placebo, **NNT = 6-8**
- side effects greater that previously published **NNH = 8**, and there is no role in acute pain
Neuromodulators, cont.

- Dizziness, edema, somnolence, and memory impairment are the main S/Es. Beware when on TCAs, or have underlying liver or kidney problems.
- **Pregabalin** benefits and harms are about the same at higher cost than gabapentin.
- But pregabalin can be dosed bid instead of qid and is easier and faster to titrate.
Neuromodulators, cont

- Topiramate has evidence for migraine prophylaxis
- Carbamazepine approved for trigeminal neuralgia, risk of SJS, (oxcarbazepine fewer S/Es)
- Lamotrigine equivocal results post-stroke
- Sodium valproate has been used for migraine prophylaxis, rash, risk of SJS
- All have been used off label for neuropathic pain
Topicals

- Lidocaine and prilocaine cream for cutaneous analgesia (+menthol for effect)
- Lidocaine 5% patches (US – PHN)
- Diclofenac gel 1% for OA, patch for MSK
- Capsaicin 8% for PHN and HIV neuropathy (do not add menthol)
- $ Shotgun: diclofenac 10%, amitriptyline, ketamine, gabapentin, lidocaine 5%
- Topicals won’t help with central NP

- 18 RCTs found up to 2008, pain >6mo
- Efficacy -0.61 SMD (-0.84 to -0.37), modest
- Altered perception OR: 4.51, NNH: 7
- Events of altered motor fxn, OR: 3.93, NNH: 5
- Events that altered cognitive fxn, OR: 4.46, NNH: 8
- “More harm than good” for general use
- More studies needed
- There may be more benefit with neuropathic pain
For Persistent Pain Consider…

- Emotional **trauma** from the injury (PTSD, etc.)
  - Psychologist for exposure therapy, EMDR etc.
  - Meditation, relaxation, mindfulness
  - +/- beta blocker, SSRI, SNRI, buspirone

- The **meaning** of the injury - catastrophe
  - Psychologist for metaphor exploration and CBT

- **Perception of disability** = best predictor of RTW
  - Pain education, hurt vs. harm, life-skills training

- **Somatization disorder**
  - Conversion disorder, factitious disorder, malingering
  - Psychiatric/psychological evaluation and treatment
Interpretation

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Chronic pain will shape behavior

- Patients tend to repeat behaviors that lead to immediate reductions in pain
  - e.g. substance use, inactivity, pain behaviors
  - Though they feel good these can increase pain and negative affect in the long run

- And avoid behaviors that lead to immediate increases in pain
  - e.g. exercise, work, drug cessation
  - Though these can hurt initially they can decrease pain and negative affect in the long term
The Bio-psycho-social-spiritual model of addiction & recovery also applies to chronic pain management.
Further References

- Drugs for pain. Treatment guidelines from The Medical Letter, vol. 8 (issue 92) April 2010