

Acetaminophen-Hydrocodone (Vicodin)

Acetaminophen-hydrocodone (brand name Vicodin) is a combination analgesic. The acetaminophen component works by reversibly inhibiting the cyclooxygenase enzymes (COX) while the hydrocodone component works as an opioid receptor agonist. This medication is indicated for moderate to severe pain. Side effects include hepatotoxicity, CNS depression, and respiratory depression. Since this medication has an opioid component, addiction and dependence may occur with chronic administration.



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Mechanism of Action

Combination Analgesic

A-nail-Jay-Z

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Opioid Receptor Agonist

Poppy-droid Recepter Dragonist

Acetaminophen-hydrocodone is a combination analgesic. It is composed of a non-opioid component (acetaminophen) as well as an opioid component (hydrocodone). Prescribing this medication instead of purely opioid analgesics helps reduce the total opioid dose and reduces risk of developing significant tolerance.

Hydrocodone

Hydrant-coding-hacker

Hydrocodone acts primarily on mu receptors, with delta and kappa receptor stimulation occurring only at relatively high doses.

Reversible Inhibition of COX

Reversing with Inhibiting-chains on Cocks

The mechanism of how acetaminophen reduces pain is not fully understood. However, many studies have shown that it may activate inhibitory descending serotonergic pathways in the spinal cord. Compared to other COX inhibitors such as aspirin or ibuprofen, acetaminophen is more active in the CNS.

Acetaminophen

A-cheetah-with-a-fin

The acetaminophen component is the non-opioid component of this combination analgesic. The addition of acetaminophen helps reduce the total prescribed opioid dose.

Indication



Moderate to Severe Pain

Moderate to Severed Pain-bolt

For acute moderate to severe pain, the lowest effective dose should be used and treatment duration should almost never last longer than 1 week. Consider non-opioid analgesics before prescribing acetaminophen-hydrocodone.

Side Effects

CNS Depression

Deflated CNS-brain

Activation of opiate receptors from the hydrocodone component causes CNS depression. The mu, delta, and kappa receptors are all G-protein coupled receptors that either cause presynaptic calcium channels to close or postsynaptic potassium channels to open, resulting in neuronal hyperpolarization and CNS depression.

Respiratory Depression

Deflated Lungs

Generalized CNS hyperpolarization from acetaminophen-hydrocodone can inhibit neurons in the respiratory centers of the brainstem. This leads to reduced drive to breathe.

Hepatotoxicity

Liver with Toxic-green-glow

Acetaminophen can accumulate in the liver and cause centrilobular hepatic necrosis. For this reason, it is advisable to keep the daily dose of acetaminophen under 7500 mg. Patients who present with acetaminophen-induced liver toxicity should be administered n-acetyl cysteine.

Considerations

Addiction

Addict in Attic

This combined analgesic's opioid component gives it a higher abuse potential. Addiction and physiologic dependence may develop with chronic use.