

Bipolar II Disorder

At least one hypomanic episode (four or more days of high or irritable mood with extra energy that never wrecked work, never caused hallucinations, never needed the hospital) PLUS at least one major depressive episode — and never a full manic episode, or it becomes bipolar I.

THE ONE-LINE TRUTH

Bipolar II is depression's great impostor: the person shows up crushed by deep sadness and never mentions the energized four-day stretches that felt like their best self — and the diagnosis lives or dies on finding those highs.

The chain — how it unfolds

1. Depression dominates the lifetime course and causes most of the disability. — why: People with bipolar II spend about 40 times more symptomatic days depressed than hypomanic across their lives — the crushing low mood, fatigue, guilt, and suicidal thoughts live in the depressive pole, not the highs.
2. Misdiagnosis as unipolar major depressive disorder — often for 5 to 10 years. — why: Patients present to doctors when depressed (the suffering phase) and don't report highs that felt good, energizing, or 'like my real self'; the average delay to correct diagnosis runs many years, and during that time treatment goes in the wrong direction.
3. Antidepressant monotherapy destabilizes the course. — why: Using an antidepressant without a mood stabilizer can trigger hypomania, mania, mixed states (depressed mood with hypomanic energy and agitation — extremely dangerous), or rapid cycling (four or more episodes per year) — a treatment-induced worsening that looks like 'treatment-resistant depression' and drives more aggressive antidepressant trials, making it worse.
4. Suicide risk equals or exceeds bipolar I, despite the 'milder' label. — why: More time spent depressed plus the occurrence of mixed states (depressed hopelessness combined with hypomanic energy and impulsivity — the deadliest combination) makes bipolar II at least as lethal as bipolar I; calling it 'milder' is a dangerous misreading that costs lives.
5. Untreated cycling erodes work, relationships, and self-trust even between full episodes. — why: The unpredictability itself — which version of me shows up this month, can I trust my own judgment, will I finish what I start — costs careers, marriages, and friendships even during stretches that don't meet full episode criteria.

6. Substance use (especially alcohol, cannabis, stimulants) becomes a common but dangerous attempt to self-regulate mood. — why: Alcohol or sedatives get used to 'turn down' hypomania or anxiety; stimulants or caffeine get used to 'lift' depression or maintain hypomanic productivity — but all worsen cycling, trigger mixed states, and add addiction on top of the mood disorder.
7. Correct treatment with mood stabilizers (lithium, lamotrigine, valproate) or atypical antipsychotics (quetiapine, lurasidone) prevents episodes and saves function. — why: Mood stabilizers reduce the frequency and intensity of both depressive and hypomanic episodes, prevent antidepressant-induced switches, lower suicide risk (lithium especially), and restore predictable function — but only if the diagnosis is made and the treatment is accepted.
8. Psychoeducation and routine (sleep, light, activity, medication adherence) become the foundation of long-term stability. — why: Understanding the illness, protecting sleep (the most powerful mood regulator — even one night of lost sleep can trigger hypomania), maintaining regular daily rhythms, tracking mood patterns, and staying on medication even when feeling well prevent relapse and preserve the gains from treatment.

Spot it — what you see, and why

- Hypomanic episodes lasting at least 4 days — feeling unusually happy, irritable, or 'sped up' with more energy than normal: The frontal and limbic parts of the brain (which control mood and drive) shift into a faster gear, driven by changes in dopamine (a brain chemical that pushes motivation and reward) and norepinephrine (which amps up alertness and energy). The mood and energy stay high most of the day, nearly every day, for at least 4 straight days — shorter than full mania but still a clear shift others can see.
- Needing much less sleep but still feeling full of energy — maybe only 3 hours instead of 8, with no tiredness the next day: The brain's sleep-wake centers (especially the suprachiasmatic nucleus in the hypothalamus, which runs the body's daily clock) are overridden by high dopamine and norepinephrine levels. The person feels charged and rested on far less sleep, which is very different from insomnia where you feel exhausted but can't fall asleep.
- Talking much more or faster than usual, jumping from topic to topic: The frontal cortex (the brain's planning and organizing center) speeds up thought production, and the normal brake on speech weakens. Words spill out quickly, ideas race ahead of the conversation, and the person may interrupt or shift topics before finishing the last one.
- Racing thoughts — ideas flying through the mind so fast it's hard to capture or finish one: Increased activity in the prefrontal and temporal cortex (areas that generate and connect ideas) floods the mind with thoughts. The brain's executive control can't slow the stream, so thoughts pile up faster than the person can sort or express them.

- Feeling unusually confident or capable — taking on big projects, making grand plans, or feeling certain about skills you normally doubt: Dopamine surge in the reward and motivation circuits (especially the ventral striatum and prefrontal cortex) inflates self-assessment and lowers normal caution. The person feels unstoppable or special, beyond what the situation supports, but not to the delusional extreme seen in full mania.
- Major depressive episodes lasting at least 2 weeks — deep sadness or losing interest in nearly everything, plus sleep trouble, appetite changes, fatigue, feelings of worthlessness, trouble concentrating, or thoughts of death: The limbic system (especially the amygdala, which flags distress, and the hippocampus, which handles memory and context) becomes overactive in negativity, while the prefrontal cortex loses its ability to balance mood. Serotonin (a brain messenger that steadies mood) and norepinephrine drop. The person must have at least 5 of the 9 core symptoms, including either low mood or loss of pleasure, nearly every day for 2 weeks or more. This depressive pole is required for bipolar II — unlike bipolar I, where it's common but not mandatory.

Red flags — act now

- Any history of a full manic episode (lasting a week or more, causing major problems at work or home, including psychosis, or requiring hospitalization) — if found, the diagnosis is bipolar I, not II, and treatment must adjust accordingly.
- Mixed state: depressive despair (hopelessness, self-blame, thoughts of death) combined with hypomanic energy (restlessness, racing thoughts, little need for sleep) — this is the single highest-risk time for suicide. Assess safety today.
- New trouble sleeping, racing thoughts, or irritability after starting an antidepressant alone (without a mood stabilizer on board) — may signal a switch into hypomania or mania. Contact the prescriber immediately.
- Suicidal thoughts, plans, or urges during any depressive episode — bipolar II depression carries equal or higher suicide risk than bipolar I. Always assess and ensure safety.
- Any new rash while taking lamotrigine — stop the medication immediately and contact the prescriber. A severe, life-threatening skin reaction called Stevens-Johnson syndrome can start as a simple rash.

Treat it — in this order

1. For bipolar II depression: start quetiapine (Seroquel) 50–300 mg at bedtime, OR lurasidone (Latuda) 20–120 mg with food, OR lamotrigine (Lamictal) titrated slowly to 200 mg daily, OR lithium titrated to blood level 0.6–1.0 mEq/L — NOT an antidepressant alone → Treats the depression (where people with bipolar II spend most of their suffering time) without triggering a switch into hypomania. Quetiapine and lurasidone have the strongest research evidence for bipolar II depression. Lamotrigine takes 6–8 weeks to titrate safely (to avoid serious rash) but works well for preventing future depressions. Lithium treats and prevents both poles and cuts suicide risk.
2. For long-term maintenance (staying well between episodes): lithium (blood level 0.6–1.0 mEq/L) OR lamotrigine 100–200 mg daily, PLUS protect a regular sleep-wake schedule (same bedtime and wake time every day), PLUS psychoeducation about the illness and early warning signs → Lithium cuts the risk of relapse into both depression and hypomania, and uniquely reduces suicide risk across all mood disorders. Lamotrigine specifically guards against the depressive relapses that dominate bipolar II (less effect on hypomanic prevention). Keeping a steady sleep rhythm is itself protective — irregular sleep is one of the strongest triggers for new episodes.
3. For a hypomanic episode: restore sleep immediately (sleep hygiene, short-term sedative if needed), remove any antidepressant, optimize the mood stabilizer dose, consider short-term quetiapine or olanzapine if mood is escalating toward mania → Most hypomanic episodes can be stopped before they flip into full mania or seed the crash into depression that usually follows. Sleep loss is both a symptom AND a driver of worsening hypomania — fixing sleep breaks the cycle.
4. Antidepressants (SSRI or bupropion) ONLY if added on top of a mood stabilizer already in place, in carefully selected patients whose depression does not fully respond to mood stabilizers alone, WITH close monitoring for switch into hypomania (watch for decreased need for sleep, racing thoughts, impulsive acts) → In some people with bipolar II, cautious addition of an antidepressant on top of lithium or lamotrigine helps lift residual depression that mood stabilizers alone did not fully resolve.
5. Psychotherapy: psychoeducation (teaching the person and family what bipolar II is, how to spot early warning signs), cognitive-behavioral therapy (CBT for bipolar disorder), interpersonal and social rhythm therapy (IPSRT — focuses on keeping daily routines and sleep-wake rhythm steady) → Regular rhythms (sleep, meals, activity), learning to recognize early signs of a new episode, and sticking with medication measurably reduce relapse rates in bipolar II. IPSRT specifically targets the rhythm disruptions that trigger episodes.

Your top priorities at the bedside

1. Screen EVERY depressed patient for lifetime hypomania before antidepressants start — why: The 4-day high hiding in the history is what separates safe treatment from a manufactured switch — this is the profession's highest-yield mood question.
2. Take bipolar II depression as seriously as bipolar I — suicide risk is equal or higher — why: 'Softer bipolar' is a myth: more time depressed plus mixed states makes this at least as lethal.
3. Guard sleep rhythm and teach it as medicine — why: Sleep-need change is both the earliest warning sign and a treatment lever in both directions.