



(CASE REPORT)



Cocaine-Induced Manic Episode and Severe Thrombocytopenia: A Complex Diagnosis of Exclusion in a Young Adult: A case Report

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International Journal of Science and Research Archive, 2025, 17(02), 701–703

Publication history: Received 08 October 2025; revised on 15 November 2025; accepted on 18 November 2025

Article DOI: <https://doi.org/10.30574/ijrsra.2025.17.2.3096>

Abstract

Background: Cocaine is a potent psychostimulant known to precipitate acute psychiatric manifestations, including manic or psychotic episodes. Hematological complications are rare but increasingly described, particularly thrombocytopenia associated with toxic or immune-mediated mechanisms.

Case Presentation: We report the case of a 32-year-old man with no psychiatric history who developed an acute manic episode following massive cocaine use. Laboratory evaluations revealed severe thrombocytopenia ($30,000/\text{mm}^3$) without hemorrhagic manifestations. Infectious, immunological, and hematological investigations were unremarkable. Cocaine-induced thrombocytopenia was diagnosed. Clinical improvement was observed under olanzapine, with full normalization of platelet count after cocaine withdrawal.

Conclusion: This case illustrates the need for rigorous diagnostic exclusion and interdisciplinary collaboration when managing psychiatric presentations associated with unexpected hematological abnormalities in cocaine users.

Keywords: Cocaine; Thrombocytopenia; Manic Episode; Levamisole; Toxicology; Hematological Complications; Drug-Induced Cytopenia

1. Introduction

Cocaine is a powerful psychostimulant made from *Erythroxylum coca*. It is widely consumed for its euphoric and stimulating effects. By blocking the reuptake of dopamine, norepinephrine, and serotonin, it can trigger acute manic or psychotic episodes. This risk exists even in individuals with no psychiatric history [1].

While its somatic complications predominantly involve the cardiovascular, neurological, and respiratory systems, hematological abnormalities—although rare—have been increasingly reported. These include severe thrombocytopenia resulting from immune, toxic, or thrombotic microangiopathic mechanisms [2,3].

The frequent contamination of cocaine with levamisole further complicates the clinical picture, as this agent is known to induce cytopenias and immune-mediated vasculitis [4].

We present a case of cocaine-induced acute mania associated with severe thrombocytopenia in a young adult, illustrating the diagnostic complexity arising from this dual presentation.

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2. Methods – Case Report Methodology

This study is a single-patient clinical case report conducted in accordance with international guidelines for case-based research (CARE Guidelines). Data were extracted from clinical records, laboratory analyses, and standard hospital evaluations performed during the patient's admission. All investigations, including hematological, immunological, and infectious testing, were conducted in accordance with institutional protocols. Patient information was anonymized to ensure confidentiality.

3. Results – Case Report

A 32-year-old man with no psychiatric or medical history was admitted to the emergency department for acute psychomotor agitation. His relatives reported marked exaltation, pressured speech, grandiose ideation, disinhibition, and significantly reduced sleep over several days. History-taking revealed massive and repeated cocaine consumption in the days preceding admission, along with occasional cannabis use.

The initial physical examination was unremarkable. Complete blood count revealed severe thrombocytopenia at $60,000/\text{mm}^3$, confirmed at $30,000/\text{mm}^3$ on citrated tube testing. No petechiae, ecchymoses, or mucosal bleeding were noted. Hemoglobin and leukocyte counts were normal.

The infectious workup (HIV, HBV, HCV, EBV), immunological tests (ANA, anti-dsDNA, antiphospholipid antibodies), and peripheral smear (absence of schistocytes) were all negative. Liver function, renal function, and coagulation studies were within normal limits.

A diagnosis of cocaine-induced thrombocytopenia was considered the most plausible explanation. Olanzapine 10 mg/day was initiated because of its efficacy in managing acute manic episodes and its favorable hematological profile. The patient showed progressive clinical stabilization with sedation, improved sleep, and reduction of psychomotor agitation. Platelet counts gradually normalized, reaching $150,000/\text{mm}^3$ on day 10 of cocaine abstinence.

4. Discussion

This case highlights the diagnostic challenges raised by acute psychiatric symptoms associated with unexplained hematological abnormalities in cocaine users. Cocaine-induced thrombocytopenia, although uncommon, is recognized in the literature. Proposed mechanisms include excessive platelet activation, direct bone marrow toxicity, or cocaine-associated thrombotic microangiopathy [3,5].

Furthermore, levamisole—frequently found as a cocaine adulterant—plays an important role in drug-related cytopenias. It is known to cause severe neutropenia, thrombocytopenia, and autoimmune manifestations such as vasculitis and agranulocytosis [6,7]. Because levamisole testing is not routinely performed in clinical settings, its contribution is often underestimated.

The rapid normalization of platelet count following cocaine cessation in this case is highly suggestive of a toxic or immune-mediated mechanism directly linked to cocaine or its adulterants. Given the broad differential diagnosis of thrombocytopenia—including infectious, autoimmune, and thrombotic etiologies—a methodical approach based on diagnostic exclusion is essential to avoid missing life-threatening conditions such as thrombotic microangiopathy or immune thrombocytopenic purpura.

Interdisciplinary management involving psychiatry, internal medicine, hematology, and clinical toxicology is crucial to ensure accurate diagnosis and appropriate therapeutic decisions. From a psychiatric standpoint, olanzapine appears to be a suitable treatment option due to its relatively safe hematological profile compared with mood stabilizers such as valproate or carbamazepine, which may worsen thrombocytopenia.

5. Conclusion

Cocaine use may lead to complex clinical presentations associated with acute psychiatric manifestations with rare but potentially severe hematological complications [2,8]. This case underscores the necessity for heightened biological vigilance when evaluating cocaine users with atypical laboratory findings.

Given the growing prevalence of levamisole-adulterated cocaine, clinicians must consider both cocaine- and adulterant-related mechanisms in the differential diagnosis.

A rigorous diagnostic approach, close interdisciplinary collaboration, and prompt discontinuation of cocaine are essential to ensure favorable outcomes. Therapeutic decisions should remain cautious to minimize iatrogenic risks, in accordance with recent evidence on hematological complications associated with both cocaine and levamisole [4, 6, 7]

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no conflicts of interest.

Statement of informed consent

The Institutional Review Board approved the study. Written informed consent was obtained from all participants before their inclusion in the study, in accordance with the Declaration of Helsinki.

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