Thrombocytopenia and Cocaine Abuse

BEVERLEY ORSER, M.D., F.R.C.P. *

This report describes a case of severe thrombocytopenia associated with cocaine abuse. Anesthetists are cautioned to rule out thrombocytopenia in patients who are known substance abusers and to consider illicit drug use in patients with unexplained low platelet counts. The etiology and management of autoimmune thrombocytopenia in drug abusers is discussed.

CASE REPORT

A 26-yr-old, approximately 70-kg man presented to hospital 12 h after sustaining a blow to the head. He was known to chronically use cannabis and cocaine. Upon examination, the man was found to be stuporous; he was able to open his eyes to command but unable to respond to verbal questions. His blood pressure was 140/80 mmHg, heart rate 70 beats per min, and respiratory rate 20 breaths per min. There was a contusion over the left occipital area; his pupils were equal and reactive but showed blurring of the optic discs; and his left arm was notably weak on flexion. Multiple skin puncture sites were noted along his forearms. Initial blood laboratory analysis was as follows: platelets 17 · 10^9/l (normal value 150–400 · 10^9/l), hemoglobin 124 g/l, white blood cells 12.6 · 10^9/l, neutrophils 81%, lymphocytes 16%, monocytes 3%, reticulocytes 150 · 10^9/l (normal value 40–100 · 10^9/l).

* Lecturer.
Received from the University of Toronto, Toronto, Ontario, Canada. Accepted for publication September 21, 1990.
Address reprint requests to Dr. Orser: Department of Anesthesia, Toronto Western Hospital, 399 Bathurst Street, Toronto, Ontario, Canada, M5T 2S8.

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A traumatic intracerebral hematoma was suspected, and 200 ml mannitol 20% was administered. The patient was transferred to our neurosurgical institute where a computerized tomography scan revealed a large right frontal hematoma, contusion of the right frontal lobe, and intraventricular hemorrhage with a marked midline shift.

The patient was given another 200 ml mannitol 20% and was transferred to the operating room, where craniotomy and evacuation of the right frontal hematoma was performed. The patient was anesthetized with isoflurane, nitrous oxide, and fentanyl. The 3-h procedure was tolerated without incident, but the surgeons noted excessive oozing from the incision. Twelve units of pooled platelets were administered prior to transfer to the neurosurgical intensive care unit. Twenty hours postoperatively, the patient regained consciousness and the trachea was extubated. Unfortunately, he sustained a marked functional neurologic deficit and was transferred to a rehabilitation center on day 30 of admission.

His platelet profile during his hospital admission was as follows: immediately posttransfusion, 185 · 10^9/l; day 1 posturgery, 65 · 10^9/l; day 14, 855 · 10^9/l; and day 26, 383 · 10^9/l. Subsequent blood analysis showed no evidence of a consumptive coagulopathy: prothrombin time, partial thromboplastin time, plasma fibrinogen, and fibrinolytic time were normal. The Venereal Disease Research Laboratory (VDRL) syphilis test and hepatitis screen were negative. Immunofluorescence for antiplatelet antibodies and circulating immune complexes were not performed. Human immunodeficiency virus antibody testing was not performed because of lack of informed patient consent.

DISCUSSION

The medical and anesthetic considerations of cocaine abuse have been reviewed previously. 


anemia in drug abusers can result from a number of causes, including hypersplenism, chronic hepatitis, bacterial or fungal sepsis, acquired immunodeficiency syndrome, and disseminated intravascular coagulation. In 1985, autoimmune thrombocytopenia in heroin addicts was first reported, and more recently, cocaine-induced thrombocytopenia was described in six habitual intranasal and intravenous abusers. These patients demonstrated a clinical course similar to acute idiopathic thrombocytopenia (ITP). They had severe thrombocytopenia, no splenomegaly, and normal bone marrow with increased megakaryocytes. Their platelet counts returned to normal after termination of drug exposure, corticosteroid therapy, and in one case, splenectomy.

The most likely cause of a low platelet count in the patient described in the current report is autoimmune thrombocytopenia. His platelet count response to transfusion was transient due to destruction of donor platelets. The thrombocytosis observed on day 14 resulted from accelerated platelet production from increased megakaryocytes, and the platelet count was normal by day 26.

In individuals who chronically self-administer opioids, a specific 7S immunoglobulin G antiplatelet antibody and circulating immune complexes have been associated with increased platelet destruction and clearance by the reticuloendothelial system. In one study, this antibody was present in 85% of drug addicts with thrombocytopenia but was not seen in patients with ITP. The stimulus for initiating the autoimmune response may be the drug or agents used as diluent. A specific antibody for quinine, a common contaminant used to dilute heroin, was reported in a patient that developed thrombocytopenia after heroin injection.

The treatment of drug-induced and idiopathic thrombocytopenia is similar. Terminating drug exposure often results in a normal platelet count in one to six weeks, but thrombocytopenia may persist for up to 3 yr. The goal of therapy is the maintenance of an adequate number of functioning platelets to achieve hemostasis. The best indicator of adequate platelet function is the bleeding time, and treatment is usually not required if the bleeding time is normal. Patients with idiopathic thrombocytopenia may have normal bleeding times with platelet counts of less than 30·10^9/l. Medical therapy includes steroids, immunosuppressant therapy, androgens, gamma globulins and plasmapheresis; splenectomy is reserved for refractory, life-threatening thrombocytopenia. Platelet transfusion is reserved for life-threatening bleeding and may not be effective because of destruction of donor platelets.

The increase in cocaine use should alert anesthesiologists to the possibility of asymptomatic thrombocytopenia in drug abusers.

REFERENCES